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ADHESIVE SPINAL ARACHNOIDITIS SIMULATING SPINAL CORD TUMOR*

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There is a group of cases with the clinical picture and course of spinal cord tumor in which, at operation, no neoplasm of the spinal cord or other pathologic change except adhesions of the arachnoid to the spinal pia and to the dura is to be found. These adhesions vary from rather fine discrete bands to adherent processes completely surrounding the spinal cord and producing complete obstruction of the subarachnoid spaces. In both varieties of adhesions marked disturbances in spinal cord function have been found, and the evolution of the clinical signs has been identical, in most respects, with that of true spinal cord tumors.

Cases of spinal arachnoid adhesions were little recognized in this clinic before the routine use of the finer cerebrospinal fluid manometric tests, which are now used in all suspected spinal cord neoplasms. It is significant that in the past three years I have encountered ten cases of spinal arachnoid adhesions, in seven of which operations were performed within the last year; whereas the records of the Neurological Institute for the thirteen preceding years have shown only two cases whose operative description would permit them to be brought into this category.

Arachnoid cysts other than cysticercus and echinococcus cysts have been described not infrequently in the literature, notably by Spiller, Musser and Martin,[1] Schlesinger,[2] Foster Kennedy,[3] Bliss,[4] Bruns,[5]

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1. Spiller, Musser and Martin: Cysts of the Spinal Cord, Univ. Penn. Med. Bull. **27**:56 (March and April) 1903.

2. Schlesinger, H.: Rückenmarks und Wirbeltumoren, Vienna, 1898.

3. Kennedy, Foster: Compression of the Spinal Cord Producing the Brown-Sequard Syndrome: Cured by Operation, J. Nerv. & Ment. Dis. **38**:689, 1911.

4. Bliss, M. A.: Cysts Within the Spinal Canal, J. A. M. A. **52**:885 (March 13) 1909.

5. Bruns: Die Geschwülste des Nervensystems, Berlin, 1908, p. 374.

Potts,[6] and in the earlier literature by Quain,[7] Calmeil,[8] Ware,[9] Wilks[10] and Browne.[11] However, a critical review of the operative records would incline one to believe that true arachnoid cysts have not been encountered. In most instances a transparent wall with fluid beneath has been encountered on opening the dura. When this wall has been opened a gush of clear fluid similar to cerebrospinal fluid has occurred, and it has been seen that the so-called cyst wall has disappeared. These descriptions from rather an early period in the history of spinal cord surgery remind one of the condition encountered when the dura is opened without opening the arachnoid. The arachnoid then appears as a distended, thin walled membrane, not unlike a cyst, from which the spinal fluid gushes when an opening is made. After it is opened, the membrane is identified with difficulty as such, lying as it does intimately on the pia and spinal cord. In a careful analysis of the reported cases of arachnoid cysts, Skoog[12] felt that only four of probable arachnoid cysts were reported, and to this number he added two of his own. The same objections hold to these as true arachnoid cysts. True cysts of the arachnoid are extremely rare, and so far as I know no case has been reported with histologic evidence of their existence. However, adhesions of the arachnoid completely surrounding the cord and walling off the cerebrospinal fluid have been reported as cysts. Thus, the terms arachnoid cysts and adhesive arachnoiditis in many instances have been used interchangeably. These are not true cysts, but are more or less complete adhesions of the arachnoid definitely circumscribed. Splendid figures are published in the reports by Feodor Krause[13] illustrating such adhesions, which have been called by him arachnoiditis adhesiva circumscripta—an excellent term, and one much to be perferred to "arachnoid cysts." Similar cases were reported by Oppenheim,[14] Spiller,[15] Munro,[16]

6. Potts, C. S.: Intradural Cysts of Spinal Meninges, J. Nerv. & Ment. Dis. **37**:621, 1910.

7. Quain: Large Cysts from the Cavity of the Arachnoid, J. Path. Soc. **6**:8, 1855.

8. Calmeil: Deux observations des kystes de la grande cavité de l'arachnoid, Gaz. d. hôp. **32**:514, 1859.

9. Ware, C. E.: Cysts from the Cavity of the Arachnoid, B. M. & S. J. **59**:259, 1858-1859.

10. Wilks: Cysts in the Cavity of the Arachnoid, J. Path. Soc. **12**:6, 1865.

11. Browne: Arachnoid Cysts, J. Psych. and Med., 1875.

12. Skoog, A. L.: Leptomeningeal Cysts, J. A. M. A. **65**:394 (July 31) 1915.

13. Krause, Feodor: Zur Kenntniss der Rückenmarkslahmungen, Verhandl. d. deutsch. Gesellsch. f. Chir. **36**:598, 1907.

14. Oppenheim, H.: Geschwülste in Bereich des cerebralen Nervensystems, Berlin, 1907.

15. Spiller: Circumscribed Serous Spinal Meningitis, Am. J. M. Sc. **137**: 95, 1909.

16. Munro: Circumscribed Serous Meningitis of the Cord, Surg. Gynec. Obst., March, 1910, p. 235.

Mendel and Adler,[17] Bruns,[18] Bonhoefer,[19] Marburg,[20] Strandgaard[21] and Sahlgren.[22] Oppenheim and Spiller have used the term circumscribed serous spinal meningitis, the French arachnöidite cloisonné, and for the less definite forms Horsley[23] has used the term chronic spinal meningitis. It would seem, then, that in the literature arachnoid cysts, arachnoiditis adhesiva circumscripta and circumscribed serous spinal meningitis, arachnöidite cloisonné and chronic spinal meningitis have all been used to describe various degrees of the same condition. I think the term arachnoiditis adhesiva circumscripta spinalis is perhaps the most satisfactory one to designate the entire group if it is supplemented by the designation arachnoiditis adhesiva diffusa spinalis, for the process may be localized or diffuse.

In none of these cases has histologic evidence been offered to show that the primary inflammatory process exists in the arachnoid. It is well known that, normally, the arachnoid is devoid of blood vessels; consequently, the term arachnoiditis would be somewhat of a misnomer unless blood vessels should have grown into the arachnoid secondarily. While this may be possible, yet in those cases of this series in which the arachnoid has been examined histologically, no evidence of vascularization has been seen and, strictly speaking, the term arachnoiditis should not be used. Possibly a larger series of cases may bring forth additional evidence to show that vessels grow into the arachnoid. It is perhaps quibbling over terms not to use the designation arachnoiditis which has already gained wide usage; hence, less confusion would occur if the term arachnoiditis adhesiva circumscripta, and with this arachnoiditis adhesiva diffusa, were used. Krause[24] has used the former term not only for the spinal cord cases, but also for those in the posterior fossa, when he and Placzuk found signs resembling cerebellar tumors, and to these he applied the term arachnoiditis cerebellaris.

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17. Mendel and Adler: Zur Kenntniss der Meningitis serosa spinalis, Berl. klin. Wchnschr. **14**:1596, 1908.

18. Bruns, L.: Ein Fall von arachnoidales Zystenbildung mit Kompression der Rückenmarks, Neurol. Centralbl. **30**:1026, 1911.

19. Bonhoefer, K.: Ueber meningeale Scheinzysten um Rückenmark, Berl. klin. Wchnsch. **11**:1015, 1915.

20. Marburg, O.: Meningitis serosa spinalis, Wien. med. Wchnschr. **69**:47, 1919.

21. Strandgaard: A Case of Meningitis Serosa Circumscripta Spinalis: Cured by Operation, Ugesk. f. Laeger **81**:2120, 1919.

22. Sahlgren, E.: A Case of Spinal Cord Compression Caused by Circumscribed Serous Meningitis, Hygiea **84**:618 (Aug. 16) 1922.

23. Horsley, Victor: Chronic Spinal Meningitis, Brit. M. J. **1**:513, 1909.

24. Placzuk and Krause: Zur Kenntniss der Umschriebenen der Arachnoiditis adhesiva cerebellaris, Berl. klin. Wchnschr. **12**:29, 1907.

Horrax,[25] in a thorough study of thirty-three cases of arachnoiditis simulating cerebellar tumor, found a thickened arachnoid and a greatly dilated posterior cistern as the essential pathologic process. Twenty-eight of the patients were followed for a period of from one to nine years, and all showed improvement or complete relief from symptoms. Since twelve of the subacute and chronic cases occurred a year after an epidemic of influenza and encephalitis, Horrax felt that there may have been some etiologic connection.

Williams [26] described a case of fifteen years' duration, and Demel [27] collected reports of forty-eight cases of circumscribed meningeal involvement which presented clinical pictures of brain tumor—sixteen in the posterior fossa. Recovery followed in approximately 90 per cent of the patients operated on. Sabatucci [28] found chronic spinal meningeal adhesions at necropsy, with a history of insidious onset which closely resembled spinal cord tumor, so that this diagnosis had been made.

Adhesions may be not only circumscribed but also multiple and extensive, extending beyond any feasible operative approach. They may vary from fine bands to extensive processes. The essential point is not only the extent of the process but also the degree with which the adhesions interfere with the normal movements of the spinal cord. By holding a definite segment of the cord fixed, while movement takes place above and below, continuous injury to the cord takes place. Normally, the spinal cord moves in a dorsoventral direction with each act of respiration. Continuous flexion of the cord at the immobile segment constitutes a low grade traumatism which eventually interferes with impulse conduction similar to that seen in abnormal angulation of the humeral condyles following fracture, where many years later signs of interference in ulnar conduction and eventually ulnar paralysis appear. Of this I shall speak later.

In Horsley's description of chronic spinal meningitis, it has been extremely difficult to determine the nature of the pathologic process. Clinically, his cases resembled closely spinal cord tumor, and were differentiated essentially by the fact that pain was widely distributed

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25. Horrax, Gilbert: Generalized Cisternal Arachnoiditis Simulating Cerebellar Tumor: Its Surgical Treatment and End-Results, Arch. Surg. **9**:95 (July) 1924.

26. Williams, M. H.: Chronic Meningitis, Lancet **2**:496 (Sept. 5) 1925.

27. Demel, R.: Die Meningitis serosa circumscripta cerebralis unter den Bilde des Hirntumors und ein Beitrag zur ihrer Aetologie, Arch. f. klin. Chir. **125**:561 (Sept.) 1923.

28. Sabatucci, F.: Chronic Meningomyelitis, Policlinico (sez. med.) **32**: 425 (Sept.) 1925.

and not confined to a definite nerve root zone. Horsley spoke of "diaphanous bands" being present, but in each case the cord appeared "shrunken and yellowish tinged." The dura was distended with fluid and did not pulsate. In the cases of this series, the spinal cord showed no such change; the dura was not always so distended with fluid, and the patients did not make such marked improvement as those in Horsley's series. Horsley believed that the process was due to gonorrhea, though only two of his patients were so infected.

ETIOLOGY

The etiologic factors are not clear. Horsley considered gonorrheal infection to be responsible. In two patients of my series, gonorrheal infection had been present. In ten of the patients, previous infectious diseases were noted, among them typhoid, influenza, encephalitis and meningitis. No history or evidence of active tuberculosis was found in any of the series. The Wassermann reaction was negative in the blood and spinal fluid of all the patients examined. It is known that many of the infectious diseases may give rise to meningeal involvement during some part of their course. During such inflammatory processes in the pia, the arachnoid could easily become adherent and the basis could thus be laid for fixation of the cord. Such meningeal involvement may perhaps be transient and in itself insignificant, though the aftermath of such abortive forms may well be adhesions such as are found in these cases. A careful history of all possible meningeal infections should be gone into in each case. Since I have had no necropsy material, it is not possible to determine whether there is any associated or primary disease of the cord coincident with the adherent arachnoid. The gross appearance of the spinal cord at operation is not characteristic. In no cases has any enlargement of the cord such as is seen in intramedullary tumors been found. A slight increase in the number of the fine arteries and veins may be seen. The color and size of the cord have been within normal limits, and its consistency, so far as can be determined by palpation, has not been altered. Nevertheless, I am unable to say that a primary disease of the spinal cord has not existed, of which the adhesive arachnoiditis is only secondary. It would be better, probably, to accept adhesive arachnoiditis as the pathologic process and let the clinical course determine whether or not an active associated primary disease of the cord is present, with the arachnoiditis as a secondary manifestation. In any event, in those patients in whom the arachnoid adhesions could be freed, recovery has ensued.

EXTENT OF ARACHNOID ADHESIONS

The extent of the arachnoid adhesions is extremely variable. In some instances small, circumscribed, definitely limited adhesions may be found, while in others a more or less extensive process may exist, reaching beyond any feasible operative relief. In one instance (J. O'N.) the arachnoid adhesion was complete and definitely circumscribed, surround-

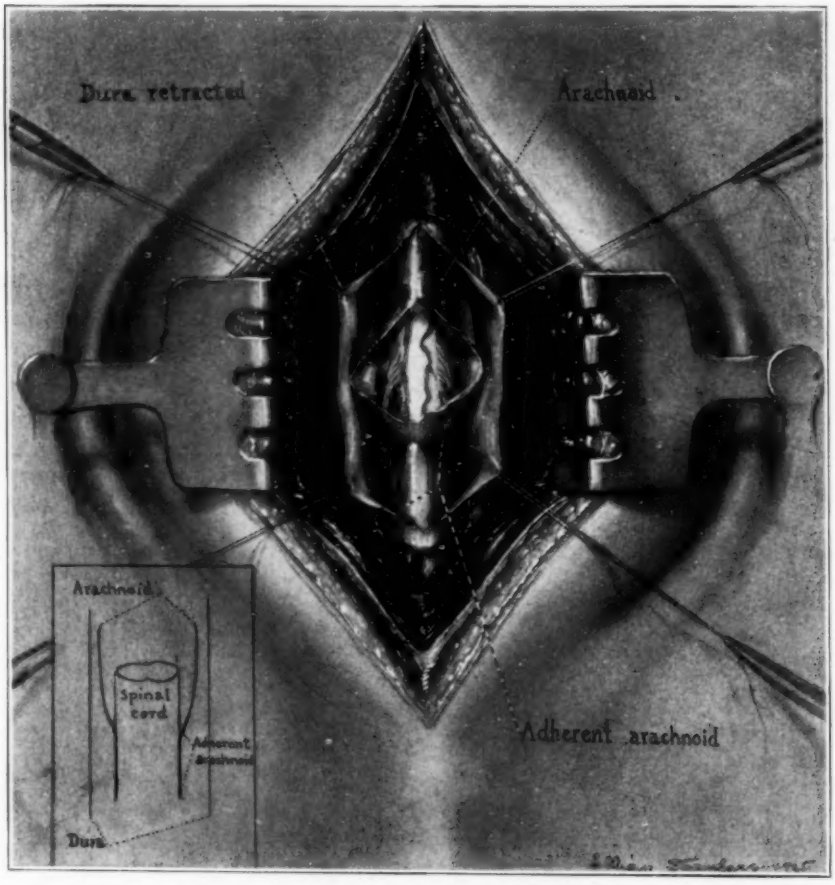

Fig. 1 (J. O'N.).—Circumscribed arachnoid adhesions completely surrounding the spinal cord and giving rise to complete obstruction of the circulation of the cerebrospinal fluid in the subarachnoid space.

ing the cord in two separate bandlike attachments separated by several segments, thus forming a definite wall enclosing cerebrospinal fluid (fig. 1). Such a condition has erroneously been called arachnoid cyst. Another patient showed unilateral adhesions—dorsal and to the left of the cord—from the second to the seventh cervical segments. A probe passed upward and downward beyond the limits of the exposure met

with resistance (fig. 2). Thus obstruction in any plane and of any extent may exist.

With such variability in the extent of the pathologic process, similar variability in the clinical picture would naturally follow, and likewise extreme variability in the operative results.

MECHANISM

When delicate adhesions were encountered as the only pathologic process in suspected spinal cord tumors, it was difficult to believe that such bands could have given rise to a symptom complex and the progres-

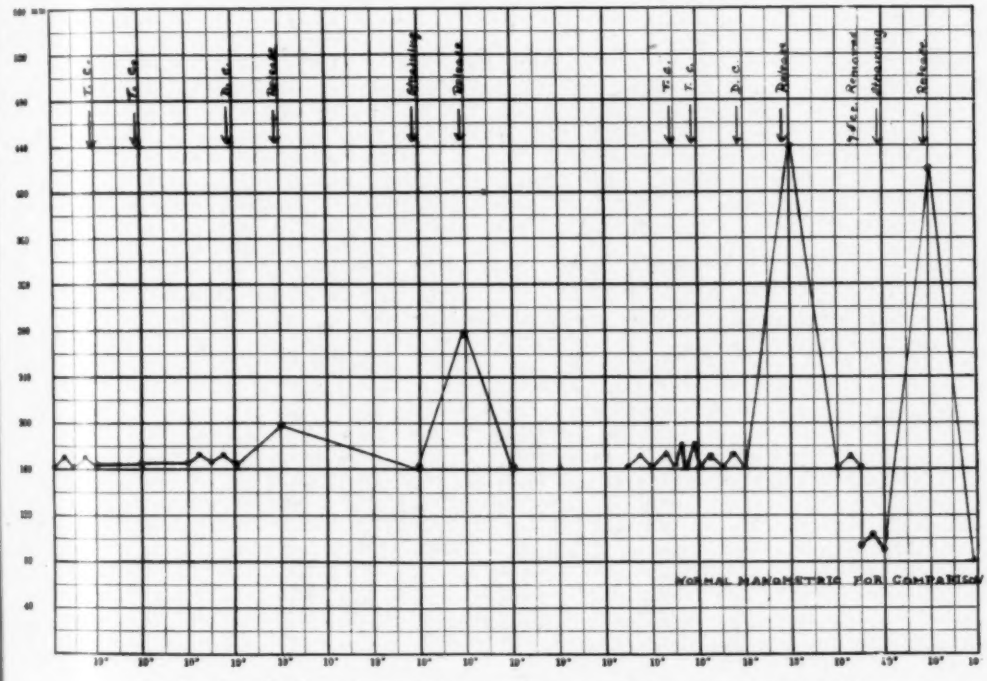

Fig. 2 (J. O'N.).—Manometric record of complete subarachnoid block compared with normal manometric chart. In this and the other charts, *T. C.* indicates touch compression of the jugular veins; *D. C.*, deep compression of the jugular veins, and 7.5 cc. removed, 7.5 cc. of cerebrospinal fluid removed with development of new level.

sive course that is usually associated with an expanding spinal cord lesion. As has been said, the importance of such adherent bands was not appreciated until their relation to the rhythmic movements of the spinal cord during respiration was understood. With each respiratory excursion, there is a dorsoventral movement of the spinal cord—an excursion of approximately 5 mm. in each direction, throughout the length of the cord, greater in the cervical and upper dorsal regions. If adherent bands are present, the cord is fixed at the points of attachment,

while above and below it moves. Thus, with each respiration, angulation at the fixed segment occurs. This in itself is perhaps trivial if of short duration, but when constant and continued over a long period of time—in some instances for years—disturbances in cord conduction can eventually occur. It is known that slight traumatism to the nervous system is well borne up to a certain point, but beyond that, in time as in degree, effects of the traumatism begin to appear and, once evident, progress steadily though slowly. An analogy to this is seen in the relation of the ulnar nerve to a fracture of the condyles of the

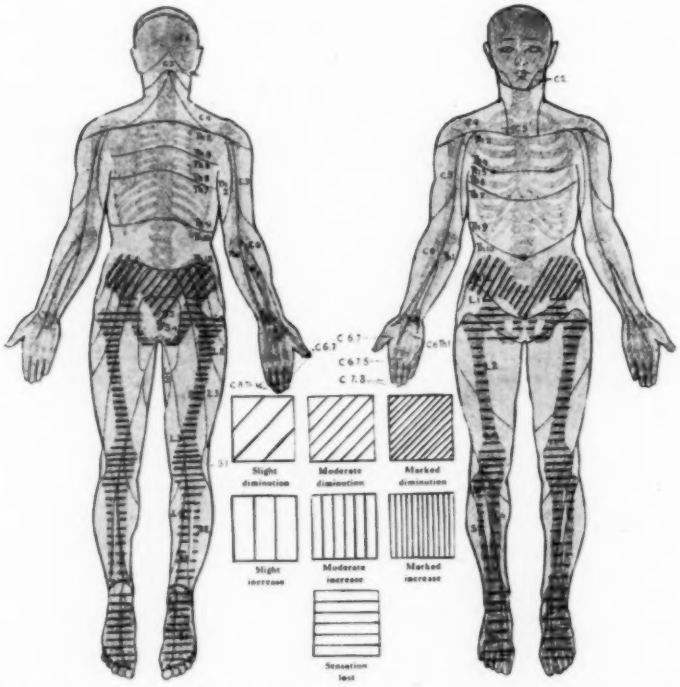

Fig. 3 (J. O'N.).—Vibratory sensation before operation, May 9, 1923.

humerus. It is known that, following such fractures with angulation, symptoms of ulnar neuritis may not occur for years—even for thirty-five or forty years after the accident—and yet at this late period signs of beginning disturbance in the conductivity of the ulnar nerve may appear and within a few years pass, with gradual increase of symptoms, to signs of complete interruption. I believe that similar mechanical factors may be at play in abnormal fixed segments of the spinal cord.

After the dura is opened and before the arachnoid is opened, these points of fixation may be seen, with movement of the spinal cord above and below them.

In addition to the dorsal and ventral movements with respiration, movements of the cord occur with movements of the trunk. This movement is so slight as to be of little consequence under ordinary circumstances. However, it is suggestive that one patient, after falling about 10 feet, developed signs of acute cord injury without signs of hemorrhage within the cord. It is conceivable that the sudden accentuation of the symptoms may have been due to the fact that the normal mobility of the spinal cord within its water bed was interfered with by the arachnoid adhesions and, consequently, a greater degree of trau-

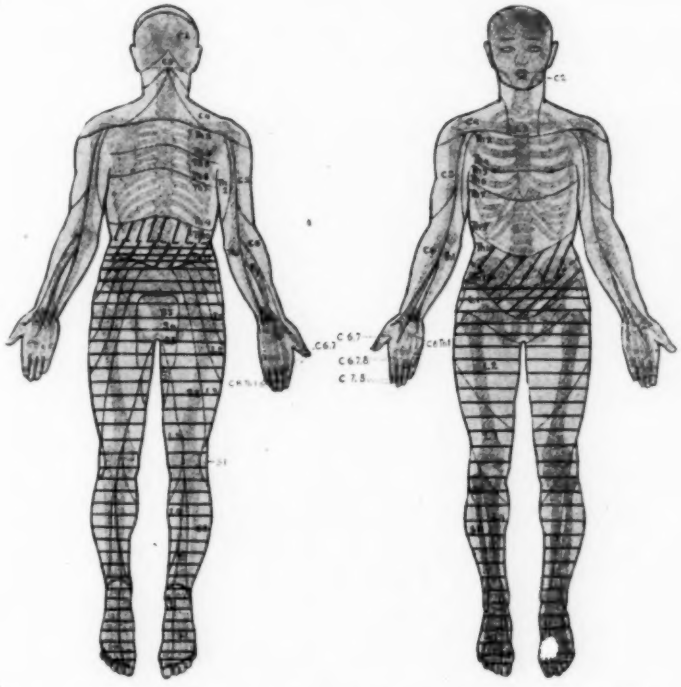

Fig. 4 (J. O'N.).—Pain and temperature sensation before operation, May 9, 1923.

matism may have been sustained by the fall than would have been the case otherwise.

OPERABILITY

Because of the variability of the character and extent of the adhesions, variability in the operative results is found. The results are dependent on the cephalocaudal extent of the adhesions rather than on their density or transverse extent. When the process is extensive and involves a large number of segments beyond a feasible exploration, the operative results are naturally poor, since adhesions are broken only in a small area and many are left, but when the adhesions are limited and

can be brought within operative approach, the results in some instances are as brilliant as those which follow cord tumors. For example, G. D. was paraplegic and bedridden, and following the operation was able to work at his business, which he has followed ever since. J. O'N., who before operation was unable to walk, now attends to her housework; and, whereas before she had a definite sensory level, no sensory changes could be found on reexamination two years after operation. Pyramidal tract signs, however, were still present, and there was some spasticity. In both of these patients the adhesive process was limited, and the results

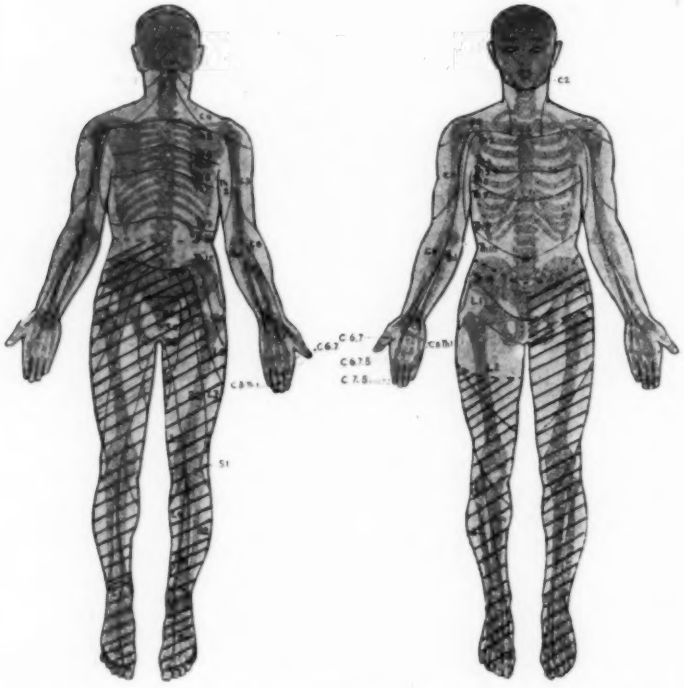

Fig. 5 (J. O'N.).—Tactile sensation before operation, May 9, 1923.

have been satisfactory. On the other hand, in . K. and C. R., the adhesive process was extensive in the c ..ual direction and consequently could not be approached, ex ..s a limited extent. In these patients, no improvement has taken place. Unfortunately, no means are at hand up to the present for differentiating the limited adhesions from the extensive. As far as the neurologic signs are concerned, both forms have been identical. In this connection, the manometric test offers no greater differentiation than the neurologic examination, and I have had no experience in these cases with lipiodol. It is possible that by a combined cistern and lumbar injection of lipiodol the

upper and lower limits might be determined. Since in some the adhesive process completely obstructs the subarachnoid space, the lipiodol would be arrested from above, and might or might not be completely arrested from below. If the diagnosis of extensive diffuse adherent arachnoiditis could be made before operation, operation would not be done, since in these cases no improvement is possible.

In most instances, adhesions are readily freed without bleeding or traumatism to the spinal cord. The absence of bleeding is further proof of the fact that blood vessels have not grown into the arachnoid. This

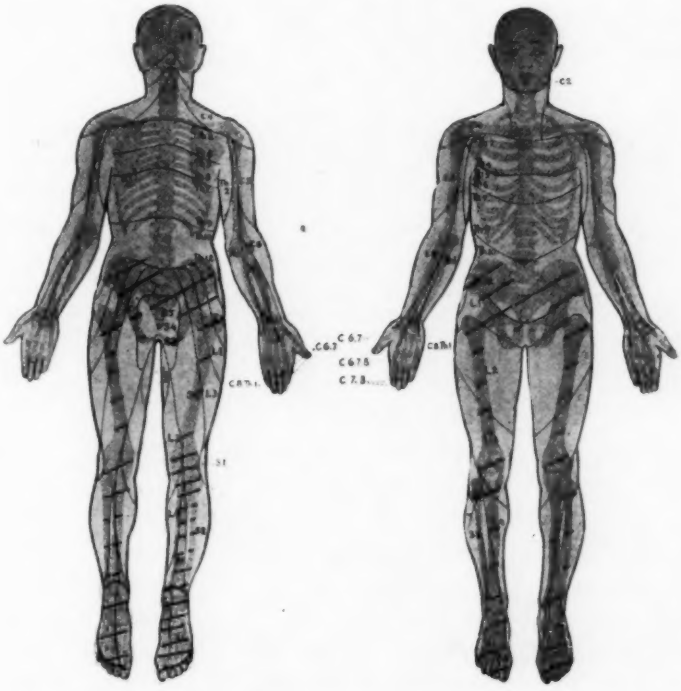

Fig. 6 (J. O'N.).—Vibratory sensation, June 24, 1923, six weeks after operation. Examination, October 15, showed complete return of vibratory sensation after operation.

is important since perfect hemostasis is obtained, the possibility of adhesions reforming is diminished.

SEGMENTAL LEVEL

The adhesive process has shown no predilection for any particular segment of the cord, though the majority would seem to be in the vicinity of the high and midcervical and midthoracic regions, the former predominating. In the cases described by Horsley, the most frequent site

of the adhesive process was in the midthoracic region. It is noteworthy that at these particular levels the dorsoventral movements of the cord are greater than in the remaining portions of the spinal cord, and consequently the traumatism which is predicated as the essential etiologic factor is more likely to affect the two regions, as shown in figures 3, 4 and 5.

TABLE 1.—*Cerebrospinal Fluid Observations in Adhesive Spinal Arachnoiditis*

Name	Manometric	Color	Cells	Globulin	Colloidal Gold	Wassermann
J. M.	Incomplete block	Clear	6	0	0———0	0
J. O'N.	Complete block	Clear	2	+	0———0	0
C. S.	Incomplete block	Clear	0	0	0———0	0
A. D.	Incomplete block	Clear	12	+	5553310000	0
J. W.		Clear	0	0	0———0	0
M. S.		Clear	4	1+	0———0	0
I. L.		Clear	0	0	0———0	0
G. D.	Complete block	Celar	5	0	0———0	0
W. M.	Nearly complete block	Clear	14	0	123310000	0
D. K.	Complete block	Clear	0	0	0———0	0
P. O'N.	Complete block	Clear	0	24 mg.* 0	222100000	0
C. R.	Almost complete block	Clear	0	31 mg.* 0	0———0	0
A. W.	Complete block	Clear	0	40 mg.* 0	0———0	0
				30 mg.*		

* Mg. refers to total protein. Unfortunately, only the globulin content and not the total protein content was determined in the earlier cases. The total protein content, according to the method of Fremont-Smith, is determined as a routine procedure in all cases at the present time.

CEREBROSPINAL FLUID

The cerebrospinal fluid, with manometric studies, has offered the most valuable differential sign that has been found. Manometric examination was made in ten of the twelve cases; of these, five showed almost complete block and the remaining five partial block. Thus, definite changes in the free circulation of the spinal fluid in the subarachnoid space were encountered. Evidence of interference with the circulation of the spinal fluid is an important factor in differentiating extrinsic from intrinsic disease of the spinal cord. It is interesting to note that in no single instance was xanthochromia present, though the manometer showed that the subarachnoid space was completely shut off in half the cases and almost completely in the remainder. The protein content was only slightly increased in two of the patients (table 1). While it is possible in spinal cord tumors not to find a marked increase in protein content in the presence of a complete or almost complete subarachnoid block, this is rare. The absence of xanthochromia and the presence of a normal protein content may be explained by the fact that tumors, whether extradural or intradural, producing sufficient compression to occlude or nearly occlude the free subarachnoid space, also interfere with the circulation of the blood supply of the cord, which is not the case in the condition considered here. Large and distended veins

on the cord are seen in spinal cord tumors. These are not found in subarachnoid block due to arachnoid adhesions, consequently xanthochromia and an increase in the protein content is not found. This point is interesting as possibly shedding some light on the production of xanthochromia and the source of the increased protein content. Xanthochromia and the high protein content have been considered by some to be due to actual products of the tumor, whereas the more usually accepted view is that both are products of transudation from the veins due to venous stasis present when block is brought about by mechanical

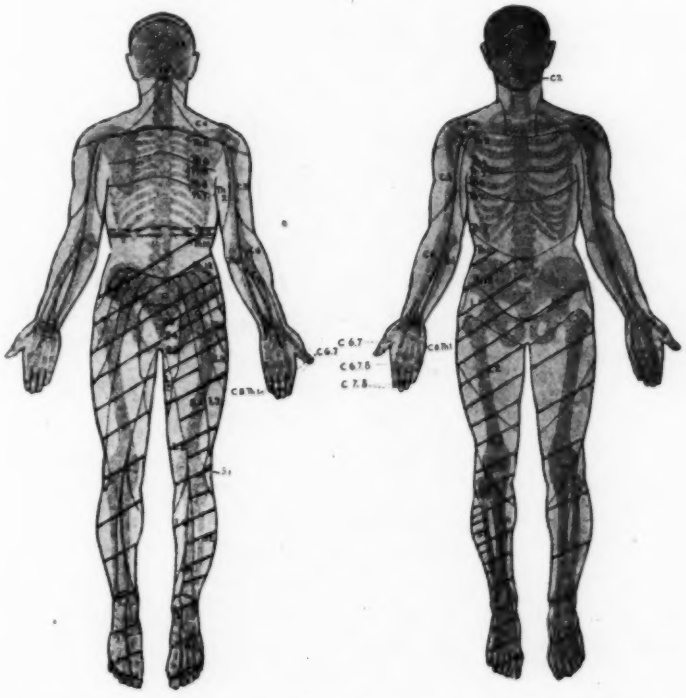

Fig. 7 (J. O'N.).—Pain sensation, June 24, 1923, six weeks after operation. October 15, five months after operation, no level could be made out. There seemed to be a slight difference between the right and left sides. Pain was not as well felt on the right as on the left.

pressure on the cord and its vascular system. In adhesive arachnoiditis, venous stasis is not present and, in spite of complete subarachnoid block neither increase in the protein content nor xanthochromia is found. Hence, obstruction of the venous circulation, with distention of the veins of the cord, apparently is essential for the production of increased total protein and xanthochromia.

The absence of increase in the total protein content is of interest, indicating that in all probability the total protein content of the peri-

vascular fluid is not greatly in excess of the cerebrospinal fluid, since the admixture of perivascular fluid with cerebrospinal fluid is far greater in subarachnoid block than occurs normally, yet no increase in the total protein content is found. Thus, in subarachnoid block due to arachnoid adhesions in which venous stasis does not occur, an almost ideal set of experimental conditions is established, in which an increase in the perivascular fluid content of the walled off cerebrospinal fluid is found. Yet this fluid, containing a higher proportion of perivascular fluid, shows no

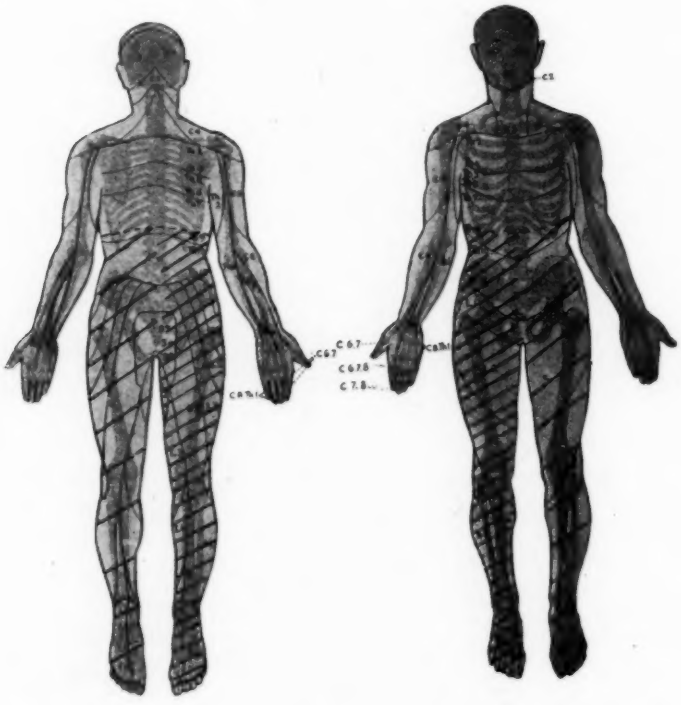

Fig. 8 (J. O'N.).—Temperature sensation, June 24, 1923, six weeks after operation. October 15, five months after operation, no differences on either side could be made out. No level could be determined.

increase in its total protein content. However, deductions cannot be too sharply drawn, since it has not been proved that the obstruction to the cerebrospinal fluid, though apparently complete as far as the manometric tests are concerned, is absolute without some seepage of the fluid from above; furthermore, it must be recalled that some absorption, even though slight, takes place along the nerve roots from that part of the subarachnoid space below the block.

SNEEZING AND COUGHING

It is known that sneezing, coughing, straining and other measures that increase intracranial, intrathoracic and intra-abdominal pressure produce a sudden, tremendous increase in the cerebrospinal fluid pressure and that the sudden impact of this force against a spinal cord tumor may cause severe radiating pains and temporary accentuation of symptoms. Similarly, sneezing and coughing in this group of patients may cause

Fig. 9 (C. R.).—Diffuse arachnoid adhesions. These extended both rostrad and caudad beyond any feasible operative approach. *AA*, adhesive arachnoid.

pain, due to the impact of the fluid against an abnormally fixed cord. Such pain was noted in two patients in this series. In one patient, sneezing produced an electric sensation down the right side of the body, and in another the pain already present was markedly increased. As suggestive evidence of involvement of the spinothalamic tract, it is interesting to observe that straining produced an electric-like sensation down the whole of one side of the body, and in this patient lateral adhesions were found, mostly on the opposite side.

VERTEBRAL TENDERNESS

Vertebral tenderness was present in half of the patients, but bore no definite relation to the level of the lesion (table 2). From the nature of the pathologic process, correspondence of vertebral tenderness to the segmental level of the lesion would hardly be expected. Thus, as a diagnostic sign, it is of little value in adhesive arachnoiditis. Likewise, in a former study of extradural tumors I found no relation between the level of the lesion and vertebral tenderness.

SENSORY CHANGES

The appearance and evolution of subjective sensory changes is almost similar to that found in spinal cord tumors. Pain has been a common symptom, occurring in eight of the twelve patients, or 75 per cent. The pain has been widespread and was referred to distal dermatomes, though what appeared to be root pains were present in three of the

TABLE 2.—*Vertebral Tenderness in Relation to Segmental Level*

Spines Tender	Segmental Level	Correspondence between Spine and Segment
Fourth thoracic	Fourth cervical	Tender spine, eight vertebrae lower than level
From the fourth to fifth thoracic.	Sixth thoracic	Tender spine from one to two vertebrae above level
Seventh cervical	Fourth cervical	Tender spine, three vertebrae below level
From the third to fourth thoracic	First thoracic	Tender spine, two vertebrae below level
Fourth, fifth and sixth cervical	Fourth thoracic	Tender spine, eight vertebrae above level
Fourth thoracic	Eighth cervical	Tender spine, three vertebrae below level

patients. Usually the pain is referred over an extensive distribution, more extensive than could be explained by involvement of a few dorsal roots. Both forms of referred pains are encountered, since the adhesions may implicate either the long fiber tracts of the spinothalamic tract or the nerve roots. Paresthesias, such as numbness, pins and needles, tingling, hot and cold and band sensations, have been present (table 3). Because the pathologic process is diffuse, it is usually impossible to correlate the presence of such paresthesias as tingling, hot and cold sensations, etc., with involvement of either the spinothalamic or the dorsal tracts. Unless a lesion is discrete and its limits well defined, it is doubtful if it is possible to determine with accuracy whether tingling or hot and cold sensations are due to irritation of the spinothalamic tracts or of the dorsal columns.

Band sensations were present in three patients. Bands were described about the ankles and knees in one, with the main pathologic process in the midthoracic region, and also in two others with the lesion principally in the cervical region. Bands about the neck were complained of by one, and about the abdomen by another. The significance of band sensation is no better understood in this group of cases than in spinal cord tumors.

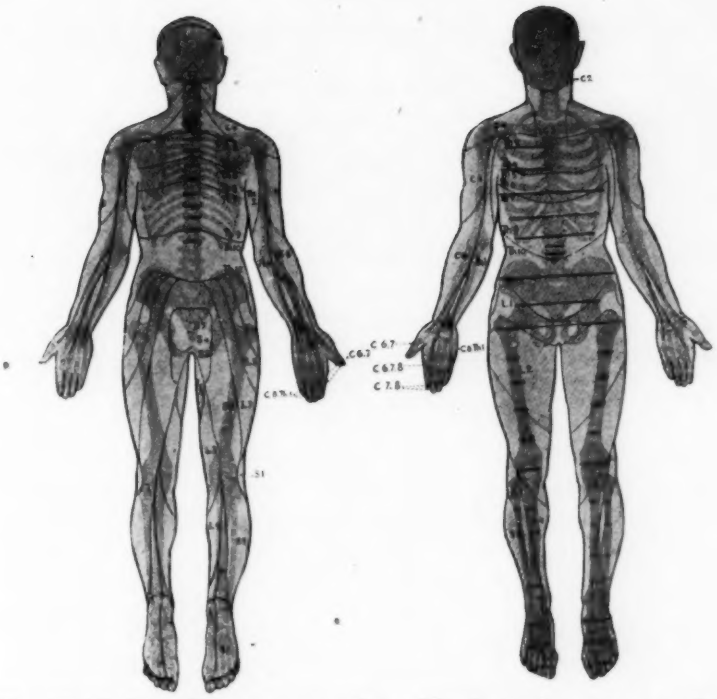

Fig. 10 (C. R.).—Vibratory sensation. Muscle tendon sense was lost in the left hand, diminished in the right thumb and index finger and lost in the fourth and fifth fingers. There was marked diminution in both legs.

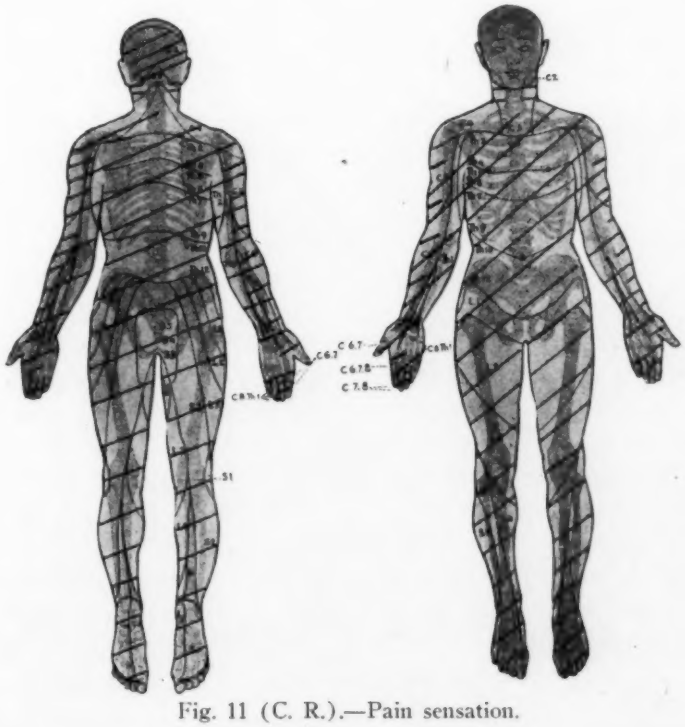

Fig. 11 (C. R.).—Pain sensation.

OBJECTIVE SENSORY CHANGES

Objective sensory changes are similar to those seen in spinal cord neoplasms. The majority of sensory disturbances correspond to the type found in extramedullary tumors rather than to those in intramedullary tumors, in that the major disturbances are not at the level of the lesion but are referred to the more distal dermatomes. Marked changes of all modalities have been found in these patients, with particular involvement of vibratory, and muscle, joint and tendon sense. The frequency with which the two are implicated is to be expected, since

TABLE 3.—*Paresthesias in Adhesive Spinal Arachnoiditis*

Name	Pain	Pins and Needles	Numbness	Tingling	Burning	Cold	Band Sensation
J. M.	Right upper and right lower extremity	Both hands	Right hand	.....	Right knee and leg	.....	.....
J. O'N. 3	0	Both feet and both upper extremities	.....	Sole	.....	.....	About ankles and knees
C. S.	0	—	—	—	.....	.....	.....
A. D.	Right lower quadrant and iliac crest	....	Right fifth finger	.....	.....	.....	.....
J. W.	Epigastric region; back of neck	....	Thighs and toes, more on left	.....	.....	.....	.....
M. S.	Back, worse on movement	.....	.....	.....	.....	.....	.....
D. K.	Neck and shoulders	Third and fourth fingers right hand	.....	.....	Lateral surface of right thigh	Right leg and left hand	About neck
I. L.	Left arm and shoulder	.....	Fingers right hand	.....	.....	.....	.....
G. D.	0	.....	Both legs	.....	.....	.....	Upper half of abdomen
W. M.	0	.....	Both legs	.....	.....	.....	.....
P.O'N.	Lower back and back of thighs	.....	.....	Right lower extremity	.....	.....	.....
C. R.	Back and right lower extremity	.....	.....	.....	.....	.....	.....

the majority of the adhesions have been dorsally placed, and consequently involve more the dorsal column functions. As in spinal cord tumors, dissociation of muscle, joint and vibratory sense is found. In three of the patients, the vibratory sense was lost, with muscle, joint and tendon sense retained. Since both modalities of sensation are carried in the dorsal columns, it seems strange that dissociation of the two modalities should exist. Such dissociation of the two forms of sensations is encountered in spinal cord tumors. Babinski pointed out that in tabes dorsalis vibratory sensation is impaired before muscle, joint and tendon sense, and that only after a considerable period is the latter

form implicated. Possibly a less degree of injury is sufficient to abolish vibratory sensation, while a more prolonged destruction is necessary to interfere with the more fundamental muscle, joint and tendon sense. Similar conditions may exist in this series, since an insidious form of traumatism of slight intensity, which only later becomes severe enough to produce marked disturbances, is being dealt with. Similar dissociations are found in the recognition of heat and cold. Often, these are not lost to the same extent, and confusion of both may exist—cold being retained many times when heat is lost and vice versa. Since both

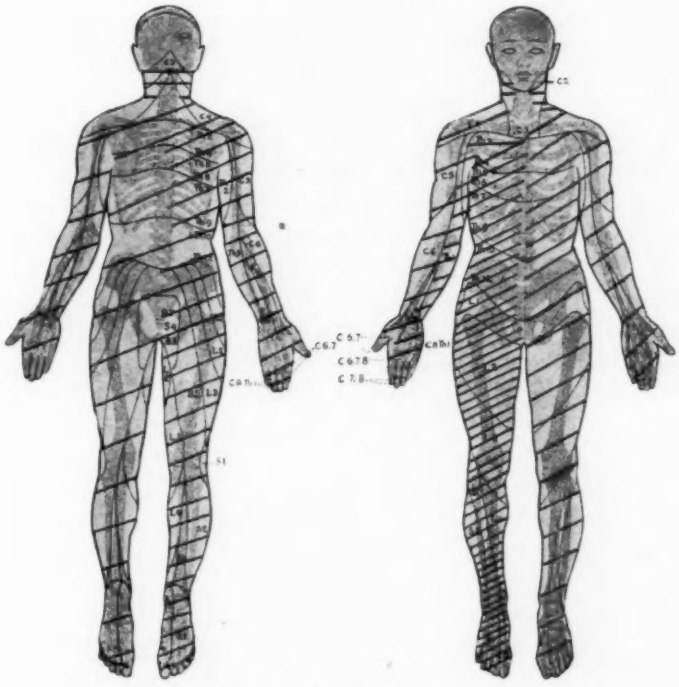

Fig. 12 (C. R.).—Tactile sensation.

of these modalities are carried in the same pathway, an equal loss of both forms might be expected. It is probable that separate fibers for these separate modalities of sensation do not exist, but rather that a difference of intensity of the stimuli is responsible for the difference in the conduction of the impulse. An analogy for this is seen in the fact that the entering fibers of the fifth nerve divide into coarse and fine fibers, the fine fibers descending to make up the spinal root of the trigeminal nerve, which Gerard [29] has shown carries pain and tempera-

29. Gerard, M. W.: Afferent Impulses of the Trigeminal Nerve, Arch. Neurol. & Psychiat. **9**:306 (March) 1923.

ture sensation. Tactile sensation is carried by the coarser branches of the same fibers which go to the principal sensory nucleus. Thus, one and the same fiber would seem to be able to conduct pain and temperature, as well as tactile sensation. This view has not been supported as yet by physiologic evidence, though it would appear to be sustained by clinical evidence.

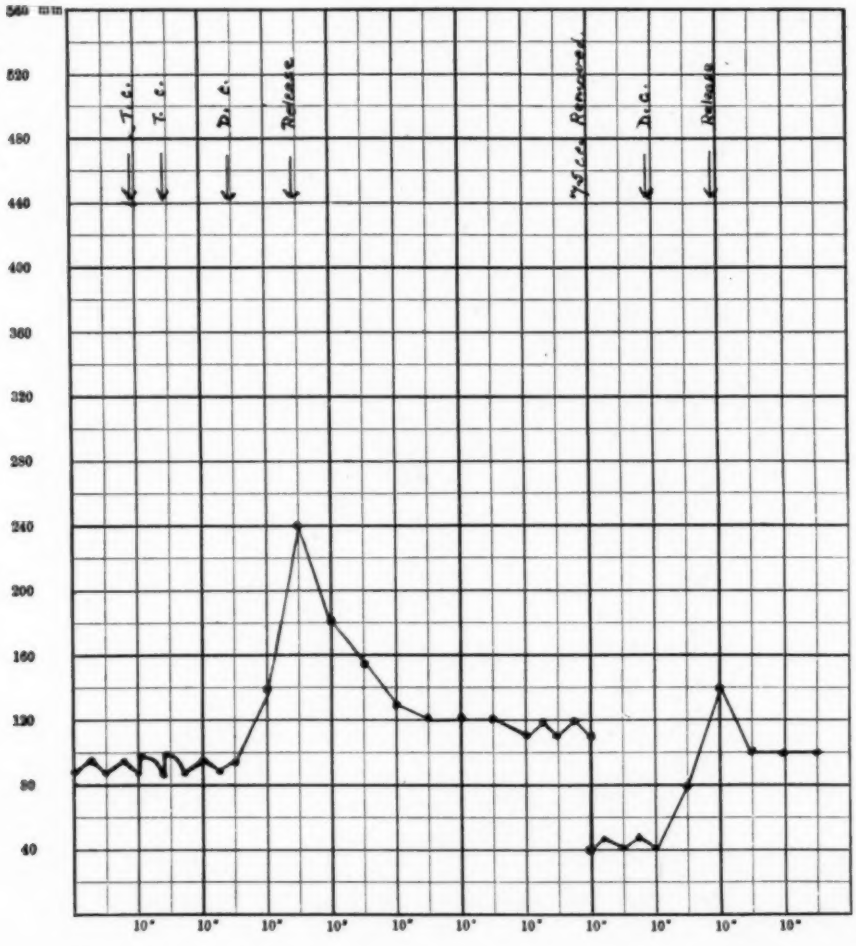

Fig. 13 (C. R.).—Cerebrospinal fluid manometric record of patient with adhesive arachnoiditis, showing partial obstruction of cerebrospinal fluid.

MOTOR DISTURBANCES

Motor disturbances have been an outstanding feature of the signs in adhesive arachnoiditis. Mass reflexes and marked exaggeration of the deep reflexes with absent abdominal reflexes, together with pathologic

reflexes, have been found in the majority. In fact, the motor disturbances have been in most instances more prominent than the sensory. The evolution of the motor signs follows closely that seen in spinal cord tumors, in that usually one lower extremity is affected first, and then the upper extremity on the same side, followed by the lower extremity on the opposite side and finally the opposite upper extremity. A most marked exaggeration of reflexes has been present in this series of cases. In one instance, a pectoral clonus and biceps clonus were present, and in another a supinator clonus and a clonus of the flexors of the fingers.

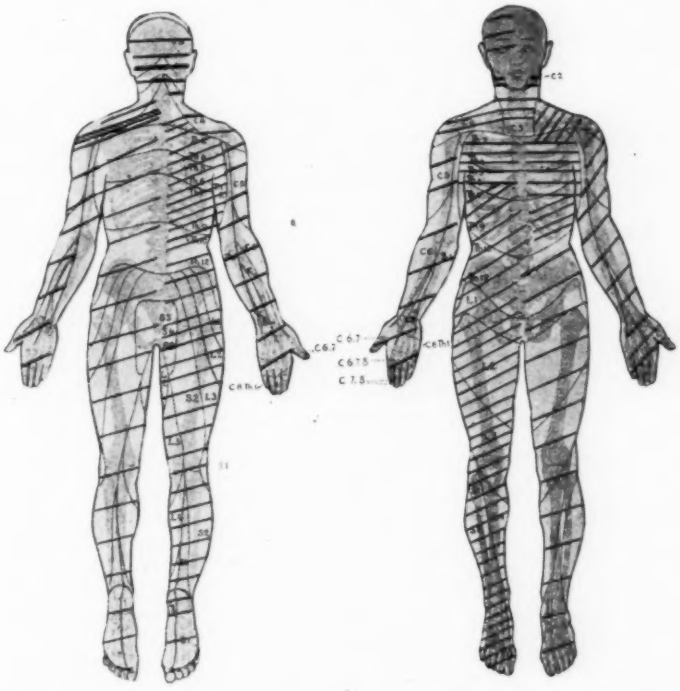

Fig. 14 (D. K.).—Pain sensation after lumbar puncture. Spinal adhesion with extension of adhesions into the posterior cranial fossa was seen. At operation, widespread adhesions in the cervical region were found, as well as extensions of the adhesive process into the posterior cranial fossa.

Atrophy of the neck muscles has been seen in one patient and atrophy of the muscles of the postaxial border of the forearm in another, closely resembling the limited distribution usually associated with tumors of one or two of the nerve roots. Fibrillary twitchings have not been observed, though they were carefully looked for. Thus I have found nothing in the motor manifestations that would enable me to differentiate adhesive arachnoiditis from spinal cord tumors.

BLADDER AND RECTAL DISTURBANCES

Bladder and rectal disturbances occur rather late in the evolution of the disease and are characterized more by dysuria and urgency than by incontinence. Incontinence of the bladder and rectum should not be expected in these patients, since the pathologic process is not extensive enough to interfere with the conduction of the rather centrally placed visceral afferent pathway which lies in and about the gray column of the cord and consequently would be affected only when all cord conduction has been interfered with. Presumably, this is a pathway of many

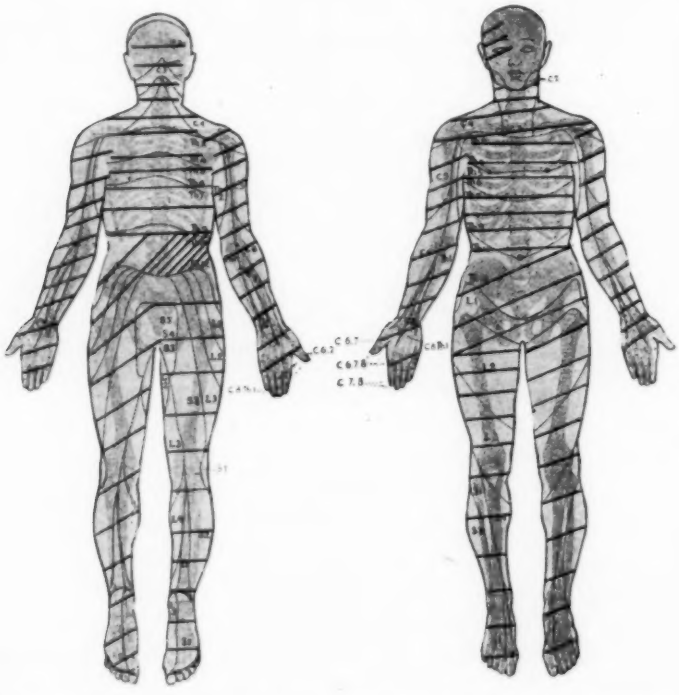

Fig. 15 (D. K.).—Temperature sensation after lumbar puncture.

neurons having a diffuse course within the spinal cord and is therefore less liable to disturbance than single, long neuron tracts. In a study of bladder and rectal disturbances in spinal cord tumors,[30] it was found that these did not appear with tumors above the lumbosacral enlargement until marked compression of the cord had occurred, sufficient to give rise to interruption of all afferent impulses. This conclusion would seem to be substantiated by the study of these cases.

30. Stookey, Bryon: A Study of Bladder and Rectal Disturbances in Spinal Cord Tumors, Arch. Neurol. & Psychiat. **10**:5 (Nov.) 1923; A Study of Extradural Tumors, Arch. Neurol. & Psychiat. **12**:663 (Dec.) 1924.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis from spinal cord tumors is extremely difficult and frequently impossible, since adhesive arachnoiditis differs in its clinical manifestations only in minutiae from those of spinal cord tumors. There are a few points which, when present, may be significant, and when considered together tend to point away from spinal cord tumor. In the

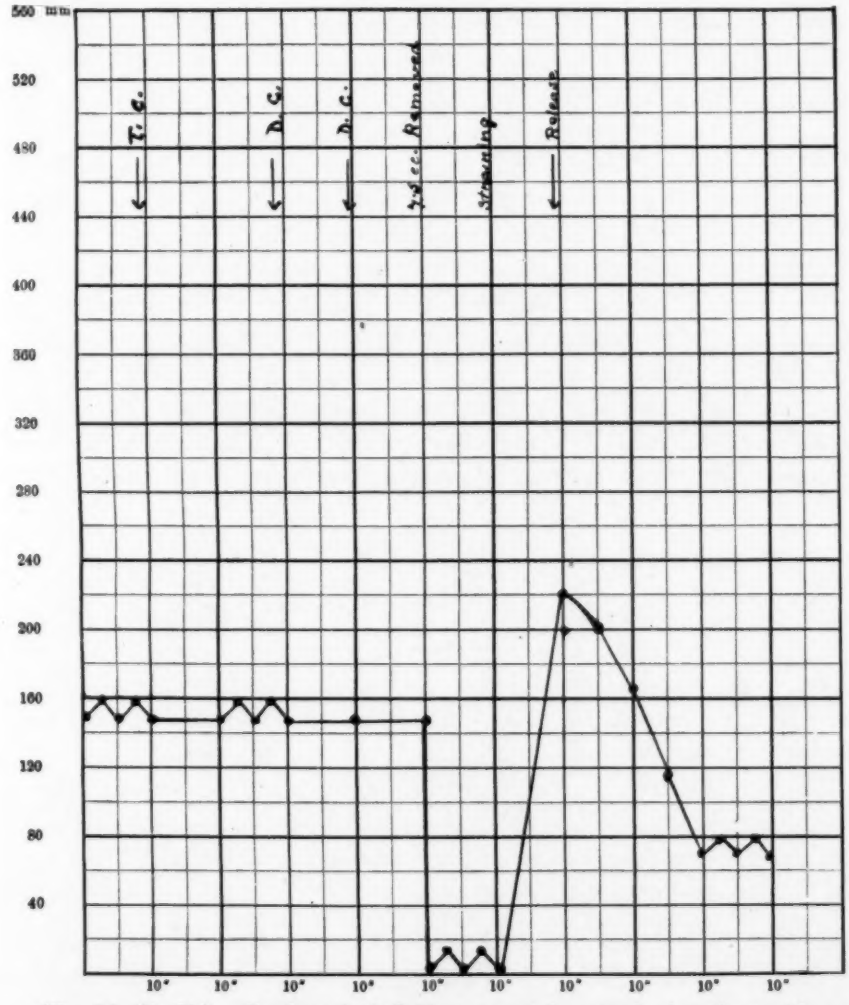

Fig. 16 (D. K.).—Cerebrospinal fluid manometric record of patient with an adhesive arachnoid, showing complete obstruction—positive in all phases.

last two cases of this series, a preoperative diagnosis of adhesive arachnoiditis was made.

The most significant point is the duration of the disease. In six, or 50 per cent, the disease had caused symptoms, on an average, for more

than nine years; in one for more than six and half years, and in another for fifteen years. In three of the patients the average time was 3.8 years and in the remainder 9.6 months (table 5). In the last two groups, the duration approximates so closely that found in spinal cord tumors that the time element could not be of differential significance. While a few spinal cord tumors have been recorded as having a history of nine and ten years' duration, this is extremely rare—so rare, in fact, that the diagnosis of spinal cord tumor might be gravely doubted.

TABLE 4.—*Presenting Symptoms in Adhesive Spinal Arachnoiditis*

Name	Age	Level	Presenting Symptom
		Cervical Group	
K.	45	From first to fourth cervical	Stiffness of neck, weakness of left arm and leg, with weakness of right upper extremity and tingling of right hand
R.	55	From fourth to sixth cervical	Difficulty in walking, pain in back; progressive
M.	45	From third to fourth cervical	Weakness of right hand, with numbness of right hand
D.	21	Fourth cervical	Pain along right iliac crest and weakness of right leg
L.	42	Fourth cervical	Weakness and stiffness of left leg followed by weakness and stiffness of left arm
M.	31	Eighth cervical	Weakness of both legs and numbness
		Thoracic Group	
D.	46	Fifth thoracic	Weakness of right leg and numbness
W.	28	Sixth thoracic	Numbness of both legs, especially left
O'N.	35	Seventh thoracic	Difficulty in walking, pain in back of thighs and in lower back
S.	53	Eighth thoracic	Weakness of both legs, right more than left
O'N.	36	Ninth thoracic	Tingling in sole of right foot and gradual weakness of right leg

TABLE 5.—*Average Duration of Disease*

Group 1		Group 2		Group 3	
Name	Duration	Name	Duration	Name	Duration
J. M.	10 years	W. M.	2.5 years	G. O.	7 months
M. S.	8.5 years	C. S.	3 years	T. W.	3 months
D. K.	8 years	J. O'N.	2.2 years	A. D.	19 months
I. L.	6.5 years				
P. O'N.	10 years				
C. R.	15 years				
Average	9.3 years	Average	2.8 years	Average	9.6 months

Another differential point is the extremely slow evolution of the symptoms. Usually in spinal cord tumors, after the symptoms first manifest themselves, their progression is rather continuous until the signs are marked. In these patients, progression has been extremely slow. I have not found in this series that pain over a wide distribution, as described by Horsley, was an outstanding symptom. While pain has existed, it often assumed a root distribution said to be characteristic of spinal cord tumors. Nevertheless, when wide pain distribution not corresponding to a root area is found, adhesive arachnoiditis should be considered, as Horsley has pointed out. In some of the patients the

pain and the muscular atrophy have been definitely limited to a distribution closely resembling that found in spinal cord tumors with nerve root involvement.

Probably the most important differential point is the cerebrospinal fluid, which shows no increase in its protein content or xanthochromia, in spite of a positive manometric test indicating subarachnoid block. An increase in the protein content is usually found in spinal neoplasms, especially when the tumor completely obstructs the subarachoid space. In these cases of adhesive arachnoiditis the protein

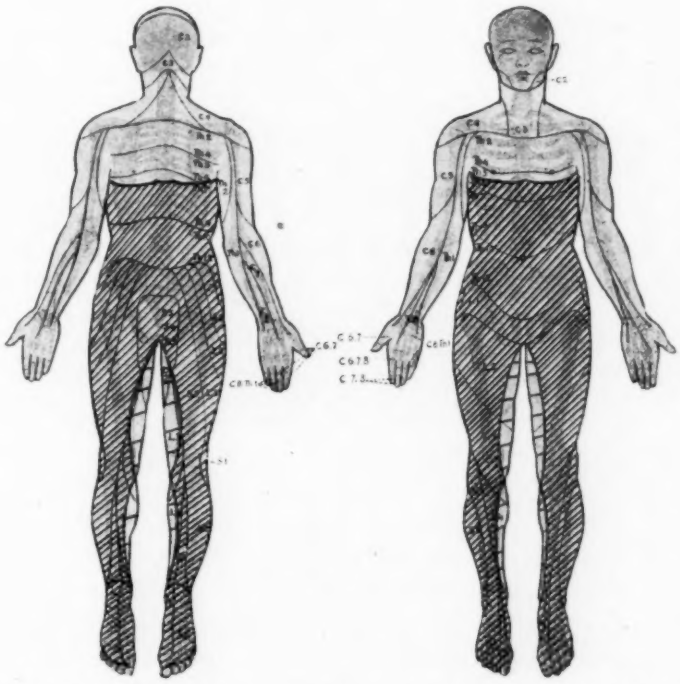

Fig. 17 (G. D.).—Tactile, temperature and pain sensation in circumscribed adhesive arachnoiditis. Though totally paraplegic and bedridden before operation, the patient showed no sensory changes or paralysis eight weeks after operation.

content has not been increased except in one or two instances, and then only slightly. The absence of xanthochromia is of less significance from a differential diagnostic standpoint, since it is not commonly seen in spinal cord tumors. With no increase in the protein content, xanthochromia would not be expected. In those cases of spinal cord neoplasms that I have seen in which xanthochromia was present, an increase in the protein content of the cerebrospinal fluid was found without exception. Thus, with marked spinal cord signs, with a long history and gradual

progression, with more or less complete subarachnoid block, without increase in the total protein content, adhesive spinal arachnoiditis should be suspected.

CONCLUSIONS

1. Adhesive spinal arachnoiditis may give rise to spinal cord signs that are differentiated with difficulty from spinal cord tumors.

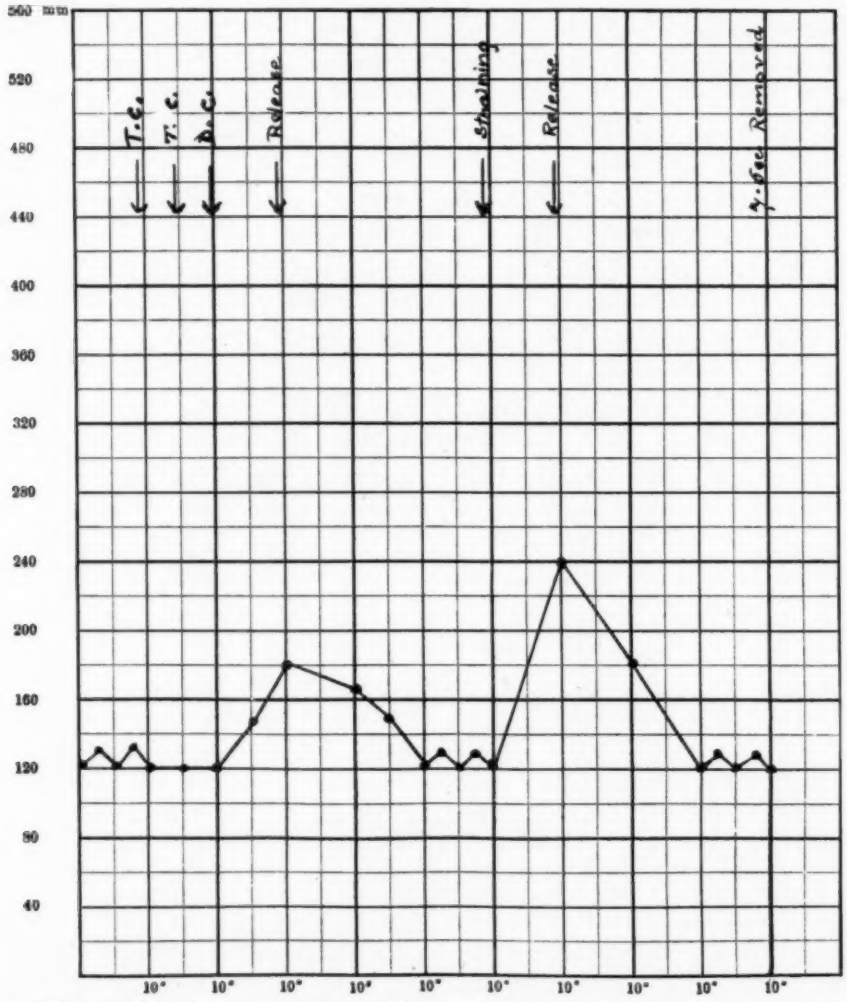

Fig. 18 (G. D.).—Cerebrospinal fluid manometric record of patient with adhesive arachnoiditis, showing complete subarachnoid block.

2. Arachnoid cysts, leptomeningeal cysts, arachnoiditis adhesiva circumscripta, circumscribed serous spinal meningitis and chronic spinal meningitis are terms used apparently to designate various degrees of the same pathologic process.

3. The terms arachnoiditis adhesiva circumscripta spinalis and arachnoiditis adhesiva diffusa spinalis are probably the best of the descriptive terms. However, since no blood vessels normally exist in the arachnoid and no evidence of ingrowth of blood vessels has been found, it is a misnomer, strictly speaking, to use the term arachnoiditis.

4. A careful investigation of all sources of possible meningeal infections should be gone into in each case as possible etiologic factors.

5. Interference with spinal cord conduction is produced by interference with the normal dorsal and ventral movements of the spinal cord during each respiration. This is a slow insidious trauma, comparable in its effects to the slow insidious paralysis that develops in the ulnar nerve after fracture of the humeral condyle. In the more complete forms of arachnoid adhesions, the weight of the impounded column of cerebrospinal fluid is an additional factor, though this is only seldom found.

6. When the adhesions are diffuse and widespread they are beyond operative approach, and consequently in the diffuse form operation is

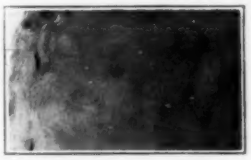

Fig. 19.—Adherent arachnoid from the cervical region. No ingrowth of blood vessels was found.

useless. But when the adhesions are circumscribed and the spinal cord is freed from them, the operative results are good.

7. Arachnoid adhesions are to be suspected when a prolonged progressive history of spinal cord signs is presented. The average duration of symptoms in half of the cases was more than nine years.

8. Manometric examination of the cerebrospinal fluid shows either complete or nearly complete subarachnoid obstruction, without, however, any increase in the total protein content.

9. Mechanism of the obstruction by adhesions without venous stasis suggests further evidence in support of the view of an increase in the total protein content of the cerebrospinal fluid in spinal cord compression as due to interference with the venous circulation with diapedesis.

10. A prolonged history, a positive cerebrospinal manometric result and a normal total protein content of the cerebrospinal fluid are the most important diagnostic points in the differential diagnosis from spinal cord neoplasms.

11. The conditions present in adhesive arachnoiditis would suggest, probably, that the total protein content of the perivascular fluid is not greatly in excess of the total protein content of the cerebrospinal fluid.

DISCUSSION

DR. M. ALLEN STARR, New York: In these cases are calcareous masses often found?

DR. BYRON STOOKEY: They are so commonly found in the presence of cord tumors and in other conditions that it has generally been thought that they had no particular significance.

DIFFERENTIAL DIAGNOSIS OF TUMORS PRIMARILY PINEAL AND PRIMARILY PONTILE*

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Intracranial tumors arising within or involving the midbrain and pons are of infrequent occurrence and often may be differentiated as to their exact site owing to the important structures which, as a rule, are involved separately and in a characteristic way in this area. There are some instances, however, in which the objective manifestations are confused or at least do not give clear-cut evidence of an exact localization. Furthermore, they are frequently mistaken for cerebellar lesions. In an effort to shed more light on such doubtful cases, a study of the tumors in this region has been undertaken. It is realized that, at the present, interest in such differentiation is largely academic, but there are three points of possible therapeutic moment to be considered. First, the pathologic type of tumor concerned may give a definite response to roentgen-ray treatment, and a more exact prognosis can then be offered. Second, tumors involving the corpora quadrigemina are almost always of pineal origin and tend to press on the midbrain rather than to invade it, so that surgical measures are at least to be considered. Third, the more accurate diagnosis of lesions in these situations might save a futile cerebellar exploration.

In Dr. Cushing's records at both the Johns Hopkins and the Peter Bent Brigham Hospitals, there have been thirty examples of tumor in the brain stem, which have been verified by necropsy. These represent, therefore, about 3 per cent of the total of somewhat more than 1,000 verified intracranial tumors in this series, and it is on this material that the present communication is based. Fifteen of the thirty tumors originated from the pineal body, and involved chiefly that portion of the midbrain just below this structure in the region of the corpora quadrigemina. Twelve of these have been reported collectively [1] from other standpoints, but will be taken up here largely in clinical comparison with the other group in the series. The other group is represented by the

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* From the Surgical Clinic of Dr. Harvey Cushing, Peter Bent Brigham Hospital.

* Read at the Fifty-Second Annual Meeting of the American Neurological Association, Atlantic City, N. J., June, 1926.

1. Horrax, Gilbert, and Bailey, P.: Tumors of the Pineal Body, Arch. Neurol. & Psychiat. **13**:423-467 (April) 1925.

remaining fifteen cases in which the tumor was largely or wholly within the pons. The two groups will hereafter be spoken of as pineal and pontile.

The sex incidence in the pineal group was eleven males and four females; in the pontile, seven males and eight females. The average age in the pineal group was 26 years and in the pontile 22 years. The average duration of time from the onset of symptoms until admission to the hospital was 9.6 months in the pineal and 13.6 months in the pontile group.

SYMPTOMATOLOGY

While many of the subjective complaints of patients in both groups were similar, or at least not definitely characteristic, there were four features that showed distinct differential criteria. Perhaps the most striking thing in this respect was the difference in the mode of onset in the two groups. All the pineal cases began with pressure symptoms (headaches in fourteen instances and failing vision in one), and the headache was said to be frontal in seven of the fourteen. Only six of the fifteen pontile cases began with general pressure manifestations. The onset in the other nine was various, but in six it was with evidence of some oculomotor disturbance such as diplopia, ptosis or strabismus.

These points are easily explained, since a pineal tumor even of small size would quickly tend to block the aqueduct of Sylvius, thus giving rise to internal hydrocephalus and immediate pressure symptoms, whereas pontile tumors would as a rule involve the cranial nerve nuclei before reaching sufficient size to occlude the cerebrospinal fluid pathway.

Hearing.—A second subjective symptom of differential value was the reference to deafness on the part of patients in the two groups. Eleven of the thirty patients complained of some acoustic difficulty, and rather unexpectedly this was true of only four in the pineal group, whereas it was recognized by seven of the fifteen patients with pontile lesions. The important feature was the way in which such difficulties were manifested, because the pineal patients did not refer the trouble to either side, while in the pontile group the acoustic loss was always definitely referred to one ear or the other. This, of course, is what would be expected from a relatively symmetrical pressure on the superior colliculi by a tumor arising from a midline structure (pineal) in the former and a unilateral peripheral involvement in the latter from a tumor that might arise at any point in the pons.

Weakness and Numbness of One Side.—In only one case of pineal tumor was there any subjective suggestion of involvement of the sensorimotor pathway, and this was manifested merely by unilateral numbness. On the other hand, eight of the patients with pontile tumors complained

of outspoken weakness or numbness (usually both) of one arm and leg, and in five instances numbness of the opposite side of the face.

Staggering Gait.—While many features of ataxia and unsteadiness were brought out in the examination of these patients, their chief complaint subjectively was staggering. This was true of five in the pineal group and of eleven in the pontile. The latter figure represents 80 per cent of the fifteen pontile tumors and would seem highly significant.

In addition to the symptoms related, there were, of course, many other subjective complaints on the part of patients in both groups. These, however, were not sufficiently constant in any great number to be of differential value. Such symptoms consisted of dizziness, vomiting, convulsions, dysarthria, tinnitus, drowsiness, etc.

OBJECTIVE NEUROLOGIC OBSERVATIONS

A. Cranial Nerves.—Olfactory: No olfactory manifestations were found.

Optic: The important differential feature respecting the optic nerves was the fact that choked disk was an early manifestation in the pineal cases and appeared late in the pontile, this being due to an early obstructive internal hydrocephalus that is characteristic of the former lesions. Despite the fact that the pineal cases from the time of their onset came under observation, on the average, four months earlier than the pontile, nevertheless, all but one of them showed papilledema, which was almost always in an advanced stage. In the pontile cases, choking was present in nine and absent or questionable in six. Ninety-three per cent of the pineal and 60 per cent of the pontile cases showed choked disks.

Oculomotor: There were many disturbances of the pupillary reactions and eye movements in both groups of cases, being present in thirteen of the pineal and a like number of the pontile.

The most important characteristic and differentiating point was the impossibility of conjugate movement upward above the horizontal plane with both eyes. This feature existed in seven of the pineal cases, while none of the pontile were lacking in this ability. This sign, when present, would seem pathognomonic of a high midbrain (presumably pineal) tumor.

Another highly characteristic feature in the pineal group was the bilateral loss of pupillary reaction to light in five of the cases in which vision, although reduced, was by no means far enough gone to produce this result. None of the patients with pontile tumors showed such a loss completely, although in one there was a unilateral fixed pupil, and in two others the pupillary reaction to light was sluggish.

Trochlear: There were no definite trochlear manifestations.

Trigeminal: In only one pineal case was there any involvement of the fifth nerve, and this was a unilateral partial sensory loss. Eleven of

the pontile cases showed definite trigeminal impairment, six being sensory, four motor and one showing both.

Abducens: No special difference was shown in the two groups with regard to weakness of the external rotators of the eye. Such weakness was present on one or both sides in eight cases of each group.

Facial: Only one pineal case showed a peripheral facial weakness, while in four others there was present a weakness of the central type. Ten of the pontile group (66.6 per cent) showed peripheral facial palsies, and in two of these the loss was bilateral.

Acoustic: As mentioned under subjective observations, the acoustic apparatus was involved in a different way in the two groups. Objectively, deafness was nearly complete and bilateral in four pineal cases and partial as well as bilateral in one more. In the pontile group, although all the patients having deafness referred it to one ear, three cases showed objectively partial bilateral impairment in hearing, while four showed only a unilateral loss. In one other pontile case, there was some questionable auditory difficulty which was not clearly defined. It is surprising that more of the pineal cases did not give evidence of acoustic loss, as all but three of the patients had large tumors which practically flattened out the corpora quadrigemina. With more perfected methods of testing the auditory apparatus, partial loss could doubtless be demonstrated.

Glossopharyngeal, Vagus and Spinal Accessory: One pineal case showed a questionable palatal weakness, while in the pontile group this was present in four cases, one being bilateral. In one other pontile case there was questionable weakness. In addition to these demonstrable features, difficulty in speech (dysarthria) was present in seven of the pontile cases, and in four there was also difficulty in swallowing. Two of the pineal cases showed these difficulties.

Hypoglossal: Three pontile cases showed involvement of the twelfth nerve or nucleus, manifested either by atrophy of the tongue on one side or by protrusion of the tongue toward one side. In none of the pineal cases was this nerve involved.

B. Hemiplegia.—Unilateral weakness was not present in any of the pineal cases. In the pontile group nine patients showed weakness of the arm and leg on one side; this was always on the side opposite a facial weakness if such was present and unilateral. In other words 60 per cent of the pontile group showed, when under observation, the classic "hemiplegia alternans."

C. Hemihypesthesia.—Two pineal cases showed generalized sensory loss over one side of the body, this being on the same side as the facial weakness of central type which was present in one patient and on the opposite side in the other. Six of the pontile cases showed a sensory loss over one side of the body, arm and leg (but not including the face).

This sensory loss was always opposite a facial weakness when the latter was present.

D. Pseudocerebellar Signs.—One or more of the usual so-called "cerebelllar" signs and symptoms (nystagmus, ataxia, Romberg sign, staggering) were present in eleven of the fifteen pineal cases and in all of the pontile. They dominated the picture to such an extent in certain instances as to cause a suboccipital exploration to be performed in four of the pineal group and in eight of the pontile. Thus 44 per cent of all these patients were subjected to operation because of uncertainty in the diagnosis. At first glance this fact appears startling, but when it is considered that there was a chance that some of the tumors might have originated in the cerebellum and extended into the pons, the only possible means of help would be from a suboccipital exploration, with sufficient tumor removal to relieve the internal hydrocephalus.

The following is a more detailed account of the various cerebellar signs presented:

1. Nystagmus: This sign was exhibited by five patients in the pineal group and was recorded as being both to the right and to the left in four instances, and as vertical in the other. Eleven, or 73 per cent, of the pontile cases showed nystagmus, it being inconspicuous in only two. In three cases it was present in all directions; in five it was both to the right and to the left; in two it was to the right only and in one it was vertical only.

2. Ataxia: In four pineal cases there was outspoken, and in one other slight, ataxia as shown in the finger-to-nose and the heel-to-knee tests. In the pontile group, ataxia was unmistakable in nine instances and in three others it was recorded as slight, making an 80 per cent incidence for the latter group.

3. Positive Romberg Sign: This was recorded in five of the pineal and in seven of the pontile cases.

4. Staggering Gait: As mentioned under subjective symptomatology, difficulty in walking was recognized by the patient in a high percentage of cases. Objectively, this was demonstrated in five of the pineal group and in six of the pontile. That it may have been present but not demonstrable in several others in both groups is highly probable, since in six of the pineal cases in which it was not recorded the patients were bedridden; this was also true of six in the pontile group, all of whom had complained of staggering subjectively.

E. Polyuria.—Curiously enough, four patients in the pineal group showed a well marked polyuria, the daily output of urine varying from 2,000 to 7,000 cc. This is easily explained in two of the patients, each of whom had a double tumor—one midbrain, and the other involving the pituitary gland. In the remaining two, the condition may have been

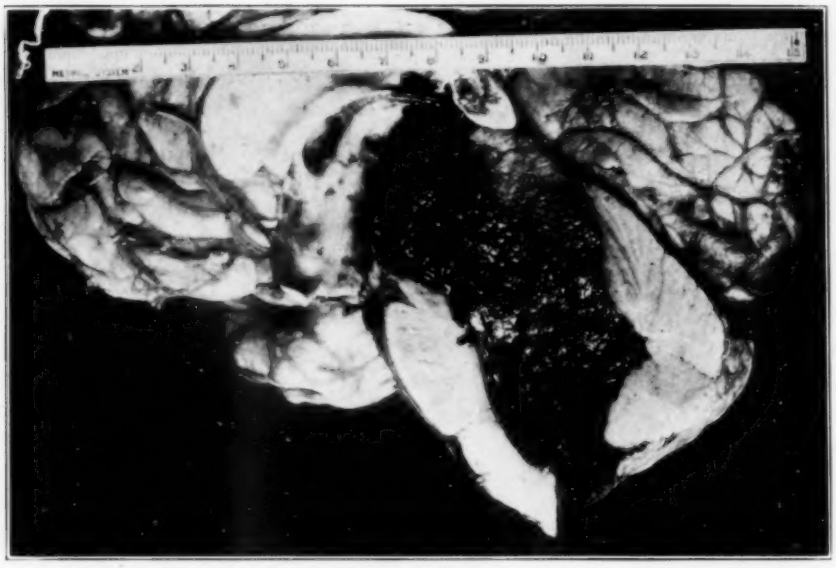

Fig. 1.—Large hemorrhagic pineal tumor projecting into third ventricle and extending downward through fourth ventricle, compressing cerebellum and brain stem. Reduced ⅓.

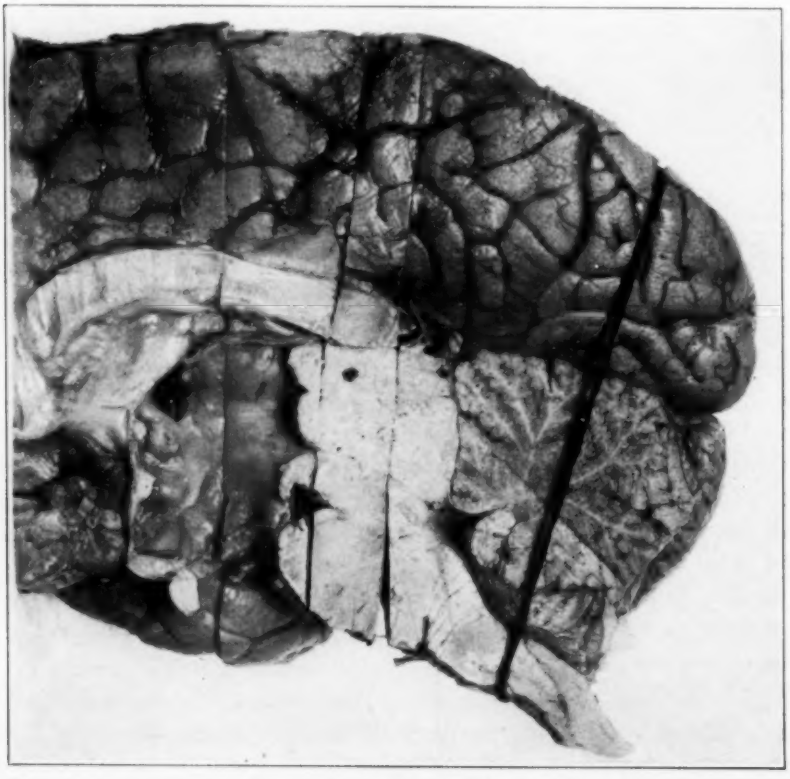

Fig. 2.—A frequent type of roughly quadrilateral, well-circumscribed pineal tumor.

due to pressure from the dilated third ventricle on the pituitary region. Polyuria was not present in any of the pontile cases.

The other metabolic disturbances which are characteristic of certain adolescent patients with pineal tumors will not be taken up here, as they have been fully discussed in a previous paper.

F. Pathology.—In the pineal group, thirteen of the fifteen tumors were pinealomas or pineoblastomas.[2] The other two were teratomas. The pathologic features of twelve of these were discussed in detail in a former communication.[1] In size they varied from that of the small bean-shaped tumor shown in figure 5 to that of the extensive growth with hemorrhage in figure 1. A considerable number of the pineal tumors, however, were roughly quadrilateral, circumscribed growths which pressed on contiguous structures without tendency to invasion. A good example of this more frequent type is present in figure 2.

One pontile tumor was a tuberculoma; all the others were gliomas. The latter were without exception of the infiltrating type, in some instances involving the pons diffusely throughout its entire extent. They were all beyond surgical reach. Examples of two different types of pontile growths are shown in figures 3 and 4.

G. Diagnosis by Ventriculography.—Two of the more recent pineal cases were studied by means of air injection into the ventricles. In one of these, the clinical diagnosis of pineal tumor was confirmed by evidence of a rounded shadow projecting into the third ventricle. An exploratory operation was performed, but the tumor was not identified. Subsequently, necropsy disclosed a tumor in the position shown by the roentgen ray, and it was seen that the operation had exposed the third ventricle anterior to the growth.

TREATMENT

In the pontile group of cases, all the tumors lay entirely within this structure, involving it diffusely, and consequently any thought of direct surgical extirpation, so far as our present knowledge goes, seems utterly futile.

The story is quite different with regard to the pineal group. As long ago as 1910, Gordon Holmes, discussing a paper by Hinds Howell,[3] commented on the fact that some pineal tumors lie detached on the corpora quadrigemina and thus might be susceptible of surgical removal.

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2. This term is now employed in accordance with the classification of Bailey, P., and Cushing, Harvey: A Classification of the Tumors of the Glioma Group on a Histogenetic Basis with a Correlated Study of Prognosis, Philadelphia, J. B. Lippincott Company, 1926.

3. Howell, Hinds: Tumors of the Pineal Body, Proc. Roy. Soc. Med. (Neurolog. Sec.) **3**:65-78, 1910.

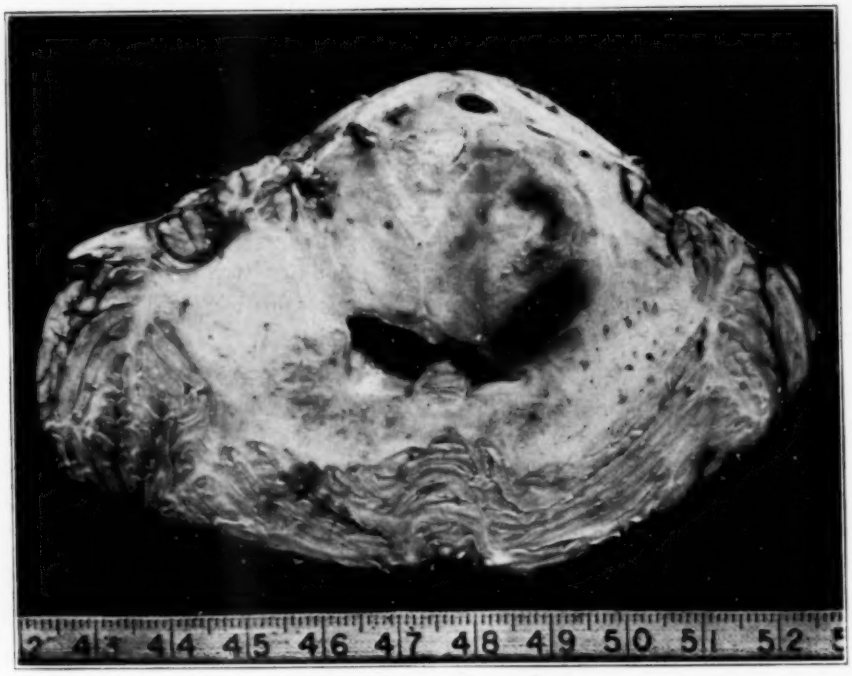

Fig. 3.—Eccentrically placed cystic glioma of pons.

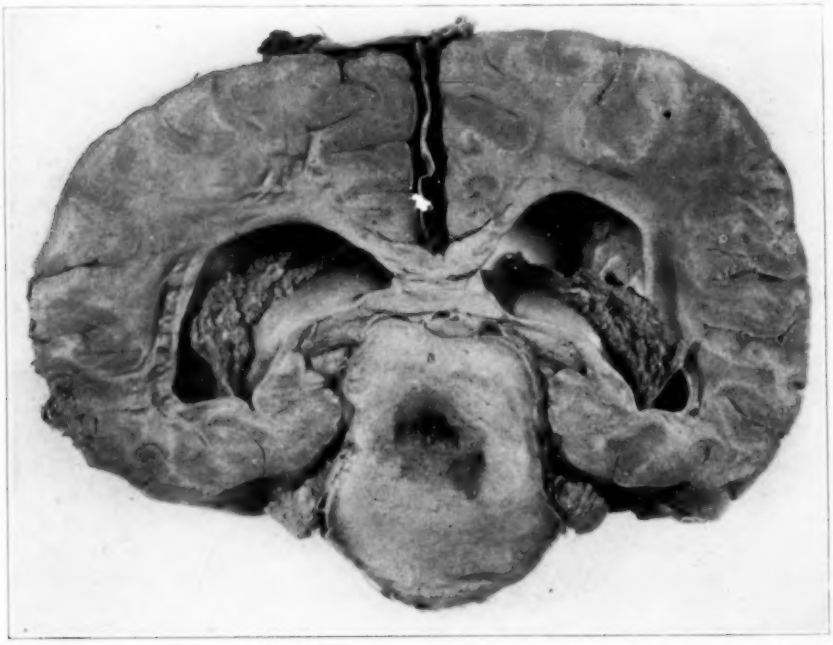

Fig. 4.—Central infiltrating glioma of pons.

In discussing the same paper, Sir Victor Horsley made the statement that a supratentorial approach to the pineal region might be feasible, and that he proposed to treat his next patient in this way.

The earliest record of an attempt to remove a pineal tumor was published by Pussep,[4] in 1914. In this instance the tumor was

Fig. 5.—Circumscribed small pinealoma compressing corpora quadrigemina. Natural size.

enucleated by an approach just above the tentorium, but the patient survived only a few hours.

The technic of a supratentorial approach for the removal of pineal tumors was published by Dandy, in 1921.[5] By retracting the posterior

4. Pussep, L.: Die operative Entfernung einer Zyste der Glandula pinealis, Neurol. Centralbl. **9**:560, 1914.

5. Dandy, W. E.: An Operation for the Removal of Pineal Tumors, Surg. Gynec. Obst. **33**:113-119 (Aug.) 1921.

half of one cerebral hemisphere away from the falx the corpus callosum is exposed, its posterior half is split, and the region of the pineal body brought into view. By this means Dr. Dandy removed two pineal tumors, one a tubercle, with survival of the patient for eight months, and the other a probable pinealoma, with death following in forty-eight hours.

These experiences are sufficiently encouraging to show that growths in this region may, in the future, be removed with success. Certainly with earlier diagnosis of pineal tumors, and with a small growth such as was present in the case here reported, the outlook is not hopeless.

COMMENT

In this study and comparison of thirty verified cases of tumors of the brain stem, it was found that there were two distinct groups: (1) those involving the upper portion of the midbrain in the region of the corpora quadrigemina, and (2) those involving the pons. The former were all tumors arising from the pineal body, and pressed on, but did not invade, the underlying structures. The latter arose within the pons and involved it diffusely.

In respect to differential criteria, the most important feature, as far as the patients' subjective complaints were concerned, was the fact that the onset in all of the pineal group began with pressure symptoms, whereas in the pontile group the first notice of trouble was usually from some oculomotor difficulty, pressure symptoms developing as a rule much later.

Objectively, the outstanding feature was the loss of associated upward movement of the eyeballs in a large number of the pineal group, this being true of none in the pontile. It is probable that all of the pineal group would have exhibited this sign, as in those in whom it was not noted the patients were either in a semistuporous condition or, in one or two instances, the records were made about twenty years ago and were not as complete as could be desired. Inactivity of the pupils to light was also an important sign, and was confined to patients in the pineal group.

Pupillary inactivity and loss of conjugate upward movement have been noted frequently in clinical reports of cases of pineal tumor, and are referred to in textbooks as being characteristic of upper midbrain lesions. In 1905, Spiller [6] discussed at length the importance of such associated eye movements, collecting reports of thirty-eight cases from the previous literature, and reporting nine personal cases. Of the total forty-seven, nineteen were verified by necropsy, and of these fourteen showed lesions involving the corpora quadrigemina. An apparent dis-

6. Spiller, W. G.: The Importance in Clinical Diagnosis of Paralysis of Associated Movements of the Eyeballs, Especially of Upward and Downward Associated Movements, J. Nerv. & Ment. Dis. **32**:417-448 and 497-530, 1905.

crepancy was that three of Spiller's own verified cases showed extensive lesions of the pons, but the loss of upward conjugate movement was probably a late manifestation from fairly complete involvement of oculomotor nuclei.[7]

In 1910, Hinds Howell [3] found that in the three verified cases of pineal tumor which he reported the pupils were inactive to light, and upward movements of the eyeballs were lost. Reviewing a large number of previously reported pineal cases, he found that the most constant ocular sign was loss of the pupillary reflex to light, and next in frequency was the loss of conjugate upward movement.

REPORT OF CASES

As a particularly good illustration of the early development of this symptomatology, one of the three cases verified since the earlier paper on pineal tumors may be quoted. This patient had a tumor no bigger than a bean, compressing the aqueduct and resting on the corpora quadrigemina.

History.—On Nov. 17, 1925, Edward R., aged 40, was referred by Dr. C. W. Rand of Los Angeles. The family and past history were irrelevant. Fifteen months before admission, he began to have headaches, which were brought on especially by coughing, laughing and straining. These headaches were usually frontal in location and became increasingly severe. The vision began to fail slightly one year before entrance, but had fallen off rapidly during the last four months. Dizziness and disturbance in memory had been noted for one year, and the patient had become drowsy and unsteady in walking during the four months prior to admission. For two weeks he had had difficulty in swallowing.

Neurologic Examination.—The patient was drowsy and apathetic. The fundi showed bilateral choked disk, with much new tissue and an elevation of from 4 to 5 diopters. Both pupils were somewhat dilated and did not react to light. Conjugate movements of the eyeballs upward or downward were impossible. There was slight ptosis of the right eyelid, and a right lower facial weakness (central type). Romberg's sign was positive, and there was moderate ataxia of both the arms and the legs by the usual tests. The deep reflexes were exaggerated throughout, with bilateral ankle clonus and positive Babinski sign. Some dysarthria and dysphagia were present.

A palliative subtemporal decompression was performed by Dr. Cushing on Nov. 20, 1925, but the patient failed rapidly, and died three days later.

Necropsy.—The brain appeared normal externally, but a median sagittal section revealed a small elliptic tumor, measuring 1.8 by 0.7 cm., just beneath the splenium of the corpus callosum (fig. 5). This tumor had completely blocked the aqueduct of Sylvius, giving rise to dilated third and lateral ventricles. The corpora quadrigemina were greatly flattened. Histologically, the growth was a pinealoma.

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7. Dr. Spiller has since stated to the author that he has come to believe that loss of associated upward movement of the eyeballs is characteristic of pineal tumors.

SUMMARY

From a review of the significant data which would serve as differential criteria in the diagnosis of tumors primarily pineal and primarily pontile, the following points would appear to have especial bearing:

1. The initial symptoms in all the pineal group were those due to increased intracranial pressure, most frequently headache, but occasionally failing vision and vomiting. Only one third of the pontile cases began in this manner. Therefore, it may be said that when the differential diagnosis between the two situations is in doubt, a history starting with pressure symptoms is strong evidence in favor of a pineal tumor; whereas conversely, the cases that begin in other ways, such as with diplopia, squint, numbness or weakness of one side, facial palsy and the like, are almost certainly pontile lesions.

2. When difficulty in hearing existed, it was always recognized subjectively as bilateral by the pineal group, and always referred to a single ear by the pontile.

3. Pressure on the sensorimotor pathway was manifested only twice, and that very slightly in the pineal group. In the pontile cases, 60 per cent presented an outspoken hemiplegia or hemihypesthesia which when present was always opposite a facial weakness if such existed.

4. Conjugate movement of the eyeballs upward above the horizontal plane was impossible in nearly one half (47 per cent) of the pineal cases. This sign, when present, would seem to be pathognomonic of a pineal tumor, as it was not present in any of the pontile group.

5. Peripheral involvement of the fifth, seventh, ninth and twelfth cranial nerves was practically absent in the pineal cases, and relatively frequent in the pontile.

6. Pseudocerebellar signs were present and outspoken in 90 per cent of the total thirty patients.

DISCUSSION

DR. W. G. SPILLER, Philadelphia: Dr. Horrax has said that in his fifteen cases of pineal tumor he had seven in which there was paralysis of upward associated movement of the eyeballs. Can he explain why he had that sign in seven and not in the other eight cases?

He also said that there was sluggishness of the pupillary reflex to light only in the cases of pineal tumor. I am not sure he meant that he found this sign in every case of pineal tumor. If not, was there any relation between those cases in which there was sluggishness to light and those in which there was the paralysis of upward associated ocular movement? Did he find in the cases with sluggishness of the light reflex any impairment of the reaction in convergence; i. e., did he find the Argyll Robertson pupil? I have come to believe that paralysis of upward associated ocular movement is almost invariably indicative of a lesion near the corpora quadrigemina.

Dr. Smith Ely Jelliffe, New York: Among the symptoms that Marburg laid stress on in his earlier description of the pineal syndrome, obesity played a large rôle. In a series of cases that Dr. Pearce Bailey and I reported, we found a preponderating group of cases in which cachexia was an objective symptom. Can anything be said about adiposity and cachexia being of particular value as localizing or as developmental symptoms?

Dr. Walter M. Kraus, New York: I have a conviction that glial tumors occur in the most newly acquired parts of the brain, phylogenetically considered. A comparison of the brain of primates with that of man, such as Brodmann has made, and a consideration of the endocranial casts of primitive men's skulls in relation to that of modern man throw light on this subject. Looking at the subject from this point of view, one would expect these tumors to begin in the prerolandic, postparietal, temporal and insular parts of the cortex, and it is precisely here that they occur. One would also expect these tumors to occur in the basal part of the pons which constitutes a recent acquisition among brain structures and is essentially a structure connecting the cortex to the cerebellum. The tegmental part is ancient and is a continuation of the medulla. One would not expect these tumors to arise in the midbrain.

Dr. Horrax has emphasized that the brain stem tumors in the midbrain region are of pineal and not of glial origin, and that those in the pontile region are glial, which coincides with my own previous deductions for these regions from this other phylogenetic point of view. The application of this idea to cerebellar gliomas is not as simple and requires further study.

I have tried to investigate this point of view of the location of glial tumors, but unfortunately the material available in my laboratory is not sufficient, and I would therefore like to hear Dr. Horrax's opinion, since he has access to such a large group of cases.

Dr. Gilbert Horrax: In regard to the question Dr. Spiller asked as to why there was not the loss of movement of the eyeballs upward in the other eight cases, I can only say that they were not recorded in the patients' histories. Some of the records, of course, went back many years, and it is conceivable that some of the patients might have shown this loss of movement which was not recorded. Furthermore, in several instances the patients were semiconscious, and therefore unable to cooperate fully in an examination.

In regard to the loss of the reaction of the pupil to light, that was true only of cases showing upper midbrain lesions, the pineal cases. None of the pontile cases showed a complete loss of the light reflex. Three of them showed a partial loss, a sluggishness to light of the pupil.

As to the question of the relation of the foregoing pupillary condition to convergence, I could not tell without looking up the records.

Answering Dr. Jelliffe's question, I have not gone into the question of the metabolic disturbances of pineal tumors in this paper, as I have discussed it elsewhere. We have had a few examples of such cases in the series. There were at least two or three of these patients with pineal tumor who showed a definite cachexia and four who showed marked polyuria, but in two instances the picture was complicated by the fact that there was also present a pituitary tumor, so that it is not shown which was playing the greater rôle. Two of the pineal cases, in which there was not a double lesion, did show polyuria and cachexia.

I do not recall definitely figures in regard to obesity, but there were, I know, at least two of the pineal cases that presented a definite though not extremely marked obesity.

DR. JELLIFFE: Precocious genital development?

DR. HORRAX: In one case there was a markedly precocious genital development, and in one other this was fairly well marked. One was a typical teratoma; the other was a pinealoma; also in a recent unrecorded case, there was a definitely precocious genital development.

As to Dr. Kraus' questions, I am afraid I cannot answer them, because I am unfamiliar with the work covering tumors in the newer parts of the brain, and I had not looked up these growths from this standpoint.

A METHOD OF MEASURING THE CEREBRAL AND CEREBELLAR CORTICAL SURFACES *

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AND
MARVIN W. DITTO, M.E.
NEW YORK

The only accurate index of the functional extent of a structure like the cerebral or cerebellar cortex is its entire surface area. The estimation of the area of the visible parts of these structures gives only a limited knowledge of their extent. Only about one third of the cerebral cortex and one fifth of the cerebellar cortex are visible. Impressed with this fact, we first made a search of the literature for methods to determine this area. The review shows that the methods used were not satisfactory, and for this reason the method here reported was devised.

REVIEW OF LITERATURE

Henneberg,[1] in 1910, undertook surface measurements of the cortex, using millimeter paper which he pressed on small pieces of the cut up brain. He was able to confirm the work of Wagner in its general conclusions and estimated that the surface of the cortex was 226,200 sq. mm., while the portion of its visible surface was 77,000 sq. mm. He further determined that the extremes in European adults were 200,000 and 248,000 sq. mm. He also showed that the left side was greater than the right and vice versa in about an equal number of persons.

Wagner [2] had previously estimated that the frontal lobe anterior to the fissure of Rolando contained 41 per cent of the surface; that the temporoparietal region contained 21 per cent and the occipital 17 per cent. Henneberg went into further detail without adding greatly to the first figures of Wagner.

Jaeger [3] approached the matter from a somewhat different point of view, using a planometric method. He estimated the gray matter as 2.5 mm. thick on the average, and the total cortical volume as 560 cc. When these figures are checked with those of Wagner and Henneberg, 222,000 sq. mm. $\times$ 2.5 mm. equals 555 c. mm.

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* Read before the Fifty-Second Annual Meeting of the American Neurological Association, at Atlantic City, N. J., June, 1926.

* From the Neuropathological Laboratory, Montefiore Hospital.

1. Henneberg: Messungen der Oberfläche der Grosshirnrinde, J. f. Psychol. u. Neurol. **17**:144, 1910-1911.

2. Wagner: Massbestimmung der Hirnoberflaeche, Göttingen, 1864.

3. Jaeger: Inhaltsberechnung der Rindensubstanz, Arch. f. Psychiat. **54**:261, 1914.

Christian Jakob [4] measured the surface of the cortex of monkeys and related his measurements to man; his conclusions are: orang-outan : man = 1.5 : 17.

Kreuzfuchs [5] is the only author whose work dealt with the surface area of the cerebellum. In 1902, he estimated this for one case only. He emphasized the great number of errors that might occur. His method was to clear with aniline oil and to draw the grossly cut cerebellum with the aid of an Edinger drawing machine. He estimated the length of the lines obtained with a curvometer. He estimated the surface to be 84,246 sq. mm., of which 16,344 (19.4 per cent) was visible. Comparing these figures with those of Wagner and Henneberg, we find that the relation of the total surface of the cortex to the total surface of the cerebellum is 2.6 : 1 in man. It is interesting in passing to quote Kreuzfuchs' estimation of the number of Purkinje cells, which he sets at 14,237,674.

From the pathologic point of view, Henneberg showed in a small number of cases that the surface is much diminished in dementia paralytica and senility, and his figures calculated for the whole brain (although they are given for one hemisphere only) are: normal. 216,442 sq. mm.; dementia paralytica, 174,288; senility, 187,326.

OUR METHOD

1. *Applied to the Brain.*—The problem in its simplest form is one of determining the area or surface of irregular spheroid bodies. The procedures followed are:

1. Serial sections are made and stained for myelin sheaths.

2. These sections are projected, enlarged and drawn on paper.

3. The accuracy of the tracing is checked from the section itself by the use of a microscope.

4. In determining the contour of a section of a small specimen or a section in which the convolutions are extremely irregular and short in length, it would be better to trace an enlargement of the section and enlarge this tracing again from five to ten times, so that the line can be distinctly followed and its length corrected by whatever the enlargement factor was originally.

5. The length of the contour or surface of the cortex, either cerebral or cerebellar, is determined by using a rotary map measuring device (fig. 1), which may or may not be attached to the movable arm of a planometer (fig. 2). The rotary map measuring device determines the

4. Jakob, C.: Von Tierhirn zu Menschenhirn, Munich, Lehman, 1911.

5. Kreuzfuchs: Die Grösse der Oberflachen des Kleinhirns, Arb. aus Neurol. Inst. Wien **9**:274, 1902.

length of the contour or outline of the cortex. Three readings of each contour are taken and averaged.

6. The essential element in the method is the instrument that makes it possible to follow accurately an irregular line and record the length of this line. This instrument is constructed of a small wheel which has a serrated edge about one-eighth inch in diameter, to which is geared by a pinion another gear driving a train of gears to an indicator. This indicator accurately measures the distance traveled by the primary wheel on the surface following the line of the paper. Its action is similar

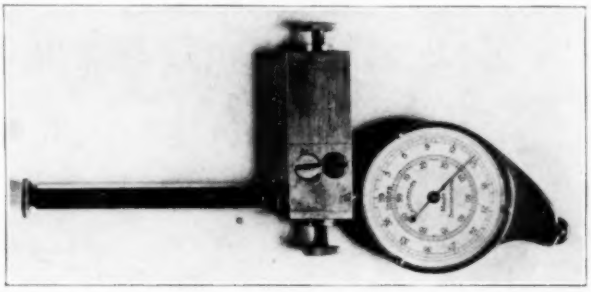

Fig. 1.—Rotary map measuring device. The metal piece above the dial with one screw at each end and two facing the reader was specially constructed so that this instrument could be attached to the planometer if desired. When it is used with the planometer more accuracy is obtained. The small wheel at the bottom of the figure is corrugated, as stated, and as it moves along the contour it turns the needle on the dial, thus permitting readings of the length of the lines.

Fig. 2.—Planometer. On the right a special attachment has been made to hold the rotary map measuring device. The rod which rises above the small handle passes through a hole in the metal attachment shown in figure 1.

to that of a speedometer, except that it disregards time. These instruments are as accurate as the problem requires, and the results are dependent on the skill of the operator using them. They are self-compensating, in that if it is necessary to retrace backward on account of a mistake, the loss is recorded and exactly the same amount of the accumulated distance is indicated by proceeding in a forward direction again. The best means of practicing the method is to attach the reproduction of the specimen to a standard drafting board to which is attached a planometer and measuring device. Dependent on the size and position of the reproduction, in some instances it might be necessary to

move the paper in order to get the lines in better position and in others to move the center point of the turntable and guide arm on the planometer.

7. The length of the contour of successive sections taken at any desired interval is considered as the upper and lower planes of a truncated pyramid (fig. 3), and the formula for calculating the area of the four sides of a trapezoid formed by truncating a pyramid is described below.

In figure 3, an irregular spheroid body corresponding to a section of the brain is represented on the right. On the left is a pyramid truncated by the plane *A B C D*. The contours *B* and *A* on the right represent the outlines of sections taken at given intervals, let us say every ten sections of a series. The lengths of these contours are determined by the instrument already described. These lengths are considered as *A B C D* and *E F G H* of the figure on the left. The

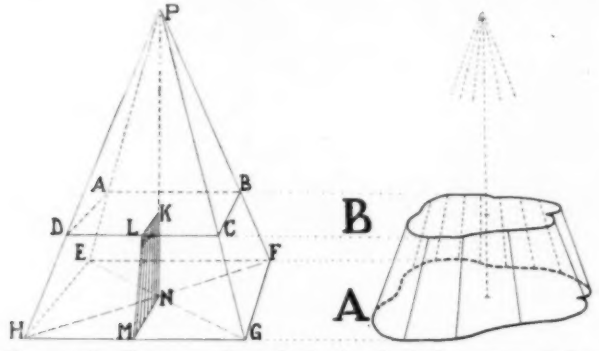

Fig. 3.—Geometric diagram of method of calculating area of four sides of a trapezoid formed by truncating a pyramid.

question then is to determine the area *D C G H* (or *C B F G*, etc.). The four sides of the truncated pyramid equal $4 \times D\ C\ G\ H$.

Let $D\ C = b$ or $\frac{B}{4}$ $H\ G = a$ or $\frac{A}{4}$.

Let $K\ N = h$, which is the distance between the sections in microns.

Let $L\ M = s$, which is the height of the trapezoid *D C G H*.

The area of the trapezoid $D\ C\ G\ H =$ its height, $L\ M \times \frac{D\ C + H\ G}{2}$.

L M is not equal to *K N*, since it is slanting, not vertical, but can easily be calculated.

$L\ K = \frac{1}{2}$ of $D\ C$ and $M\ N = \frac{1}{2}$ of $H\ G$.

K N is known.

L M is calculated as follows:

$(L\ M)^2 = K\ N^2 + (M\ N - K\ L)^2$.

Converting this into the letters used above as the equivalents:

(1) $s^2 = h^2 + \left(\frac{a - b}{2}\right)^2 = \frac{4h^2 + (a - b)^2}{4}$.

(2) $s = \frac{1}{2} \sqrt{4h^2 + (a - b)^2}$. Substituting s in the formula $s \times \frac{a + b}{2}$ for the area of the trapezoid *C D H G*, it is found that the formula for this area is

(3) $\frac{1}{2} \sqrt{4h^2 + (a - b)^2} \times \frac{a + b}{2}$. Multiplying this by four the formula is

(4) $(a + b) \sqrt{4h^2 + (a - b)^2}$.

8. The uppermost and lowermost sections of the specimen also have an area. This is determined planometrically and is added to the results of the other method.

2. *Applied to an Endocranial Cast.*—In order to determine the surface area of an endocranial cast, it is necessary to obtain a piece mold of it; that is, a mold which can be taken apart in such a manner as not to destroy the cast made within it due to undercutting. The piece mold made of plaster of Paris is properly soaked in water until the air is all driven out. It is then filled with a mixture of pure beeswax and paraffin to which is added a small amount of petrolatum to keep the mixture slightly soft. The wax is poured into the mold. The wax cast is then cut with a long, thin, sharp knife at measured intervals and its surface is determined, as already described for brain tissue.

DISCUSSION

Dr. Sidney I. Schwab, St. Louis: What are the results in relation to the older estimations?

Dr. Walter M. Kraus: We have not as yet applied this to the human brain, but we have applied it to the guinea-pig's brain. We have found that in this animal the surface area of the cortex is about 4,000 square millimeters. We are now working on the human brain and hope to carry out that estimation this year.

INVOLVEMENT OF CENTRAL NERVOUS SYSTEM SECONDARY TO PRIMARY CARCINOMA OF LUNG

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ROCHESTER, MINN.

The neurologist, whose interests are concentrated on diseases of the central nervous system, is likely to overlook the influence of a primary source of trouble elsewhere in the body. When this primary source remains latent, so far as signs of its presence are concerned, the possibilities of error are even greater. Primary carcinoma of the lung is not a common disease and therefore not often thought of, especially in connection with its distant influence on the nervous system. Ewing [1] estimated that about 1 per cent of all carcinomas were of this region, and in necropsy examination of 16,578 cases of carcinoma from various sources, 168 cases of carcinoma of the lung were recorded.[2] Barron [3] recently gave a much higher figure, 9 per cent, and both from his own experience and from a study of the statistics of the last forty years he inferred that the disease was becoming more common.

Grossly, the carcinomatous tumors of the lung may be classified as (1) nodular, (2) diffuse or lobar and (3) infiltrating. The third type is the most common, and the second type the least common. The nodular type of tumor is circumscribed, grayish-white or yellowish, and occasionally shows areas of necrosis and small cavities. The diffuse or lobar type sometimes grossly resembles lobar pneumonia. The one or several lobes involved are solid grayish, suggesting the gray hepatization of lobar pneumonia. The infiltrating variety generally has its origin in one of the larger bronchi and infiltrates the surrounding lung parenchyma; sometimes the tumor follows closely along the bronchial ramifications. There may be occlusion of a bronchus from compression or from the filling of its lumen by the growth. In advanced stages, a main tumor surrounds the hilum and large bronchi. Secondary changes in the form of bronchiectasis or atelectasis may occur, and putrefaction of retained mucus, pus and tumor detritus often excites fatal gangrene. Large vessels may become eroded and pulmonary hemorrhage result.

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* From the Section on Neurology, Mayo Clinic.

* Read before the Chicago Neurological Society, April 15, 1926.

1. Ewing, James: Neoplastic Diseases, Philadelphia, W. B. Saunders Company, 1919.

2. Adler, I.: Primary Malignant Growths of Lungs and Bronchi, New York, Longmans, Green and Co., 1912.

3. Barron, Moses: Carcinoma of the Lung: A Study of Its Incidence, Pathology and Relative Importance, Arch. Surg. **4**:624 (May) 1922.

The mass may infiltrate the mediastinum, pericardium, auricles and great veins. Extension to the pleura is frequent, and serous or bloody pleural effusion occurs in nearly 50 per cent of cases.

METHOD OF GROWTH

The metastatic growths of pulmonary carcinoma are usually numerous and widespread, but they vary greatly with the grade of malignancy. On account of the great vascularity of the lung and the frequent invasion of blood vessels by the tumor, the facility of dissemination is obvious. The ease with which tumor cells can be carried by the pulmonary veins to the left side of the heart and thence to the general circulation makes carcinoma of the lung one of the prominent sources of metastasis to the brain and other organs. The abundant lymph channels lead to infection of the bronchial nodes in a high proportion of cases, and to early extension throughout the lung and pleura. Extensions to the cervical lymph nodes and to the lymphatic spaces of the neck are common. Hassin [4] has suggested that from there, by a retrograde ascent of the perineural lymphatics of the cranial nerves, the subdural and subarachnoid spaces may be reached and invasion of the cerebral meninges may thus take place. Another path of spread for the cancer is along the lumen of the bronchi by replacement of the mucosa. The periarterial and peribronchial lymphatics frequently provide the pathway for further progress. The organs involved by metastasis vary in frequency. In 374 cases, Adler reports invasion of the bronchial nodes in 117, the mediastinal nodes in forty-five, the tracheal in twenty-six, the cervical in twenty-three, the retroperitoneal in twenty-three, and those in many other regions less frequently. The liver was invaded in 103 cases, the pleura in fifty-two, the pericardium in thirty-nine, the heart in thirty, the kidney in fifty-eight, the suprarenals in thirty-eight, the spleen in eighteen and the brain in fifty-three. The bones were involved fifty-seven times and often extensively, the ribs, spine, skull and sternum suffering in the order named. In thirty-three cases there was no metastasis. Dosquet found that in 105 cases of carcinoma of the lung the central nervous system was involved in 31.4 per cent.

SYMPTOMS

The physical signs and symptoms vary according to the extent of the pulmonary carcinoma and its metastasis at any given time in the course of the disease. Clinically, the cases may be divided into three groups: (1) Those in which the clinical signs and symptoms are

4. Hassin, G. B.: Histopathology of Carcinoma of the Cerebral Meninges, Arch. Neurol. & Psychiat. **1**:705 (June) 1919. Hassin, G. B., and Singer, H. D.: Histopathology of Cerebral Carcinoma, Arch. Neurol. & Psychiat. **8**:155 (Aug.) 1922.

solely those of pulmonary disease with no evidence of metastasis; (2) those in which, in addition to signs of the primary tumor growth, there are also signs indicating the involvement of other organs by metastasis, and (3) those in which metastasis occurs early and produces the dominant signs and symptoms with few or no signs indicating the primary source of the disease. In the last group the central nervous system is frequently involved early, and the clinical picture at one or the other stage of the disease may be solely that of disturbance of function in the brain, cord, meninges or nerve roots. It is this group of phenomena that is of chief interest in this paper.

REPORT OF CASES

CASE 1.—A woman, aged 40, was brought to the clinic, Oct. 14, 1919, because of headaches and mental deterioration. She had had mild headaches all her life, but for twelve months these had been more severe and were felt chiefly in the frontal region. Associated with these headaches were attacks of forcible and sudden vomiting. Four months before, she had complained of attacks of dizziness lasting about three hours and occurring every three weeks. In one of these attacks, speech had been lost for about an hour. For months, she had complained of a dull, continuous ache over the right upper quadrant of the abdomen with acute exacerbations. Two weeks before her visit to the clinic, she had experienced for the second time sudden loss of speech, accompanied this time by weakness of the right arm and right side of the face. These symptoms passed off within an hour, but she had become dull and drowsy thereafter. While her memory had been failing for a year, at this time it failed almost completely.

She was dull and apathetic, with defective memory, and was sometimes dissociated as to time, place and person. The mental status, however, fluctuated from day to day, but at no time was she at all bright. She was listless, yawned considerably and slept most of the time while under observation. Loud, long râles were heard over the right side of the chest near the median line and at the level of the root of the lung. Otherwise, there were no signs of pulmonary disease. A roentgenogram of the head showed nothing unusual. There was slight edema of the optic disks, not marked enough to be measured. Spinal fluid examination showed five small lymphocytes. There was some weakness of the right arm and right side of the face and a slight tremor on performing the finger-to-nose test with the right hand. There was a suggestion of Babinski's sign on the right side. The temperature and pulse remained normal until just before death. A diagnosis was made of primary cerebral tumor, left frontal lobe. The patient became more dull and stuporous and finally, October 31, left subtemporal decompression was performed with exploration of the left cerebral cortex. At operation nothing of importance was seen, except resistance to the passage of a trochar in different parts of the cerebral substance. She died, Nov. 2, 1919.

The diagnosis at necropsy was: (1) primary carcinoma of the left lung: (2) metastatic carcinoma of the tracheal and bronchial lymph nodes; (3) metastatic carcinoma of the liver; (4) multiple areas of metastatic carcinoma in the brain with hyperemia of the pia mater and flattening of the convolutions. The pulmonary carcinoma was chiefly around the hilum and primary bronchi, and extensions into the parenchyma of the lung along the vessels and bronchi were plainly visible. There were about seven or eight grossly visible cerebral metastatic tumors. The largest measured 5 cm. in diameter, the smallest 3 cm.

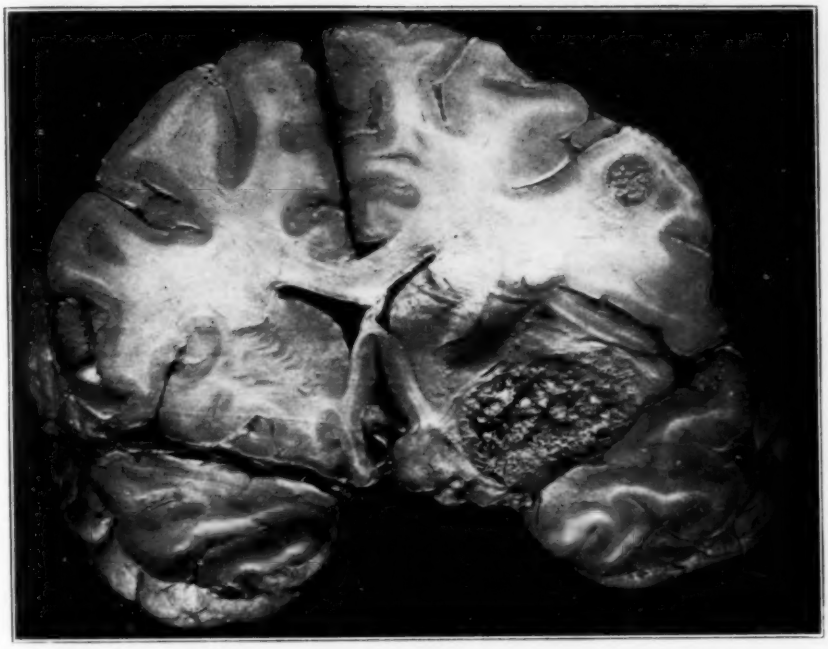

Fig. 1.—Coronal section of cerebral hemispheres showing multiple areas of metastasis from primary carcinoma of the lung.

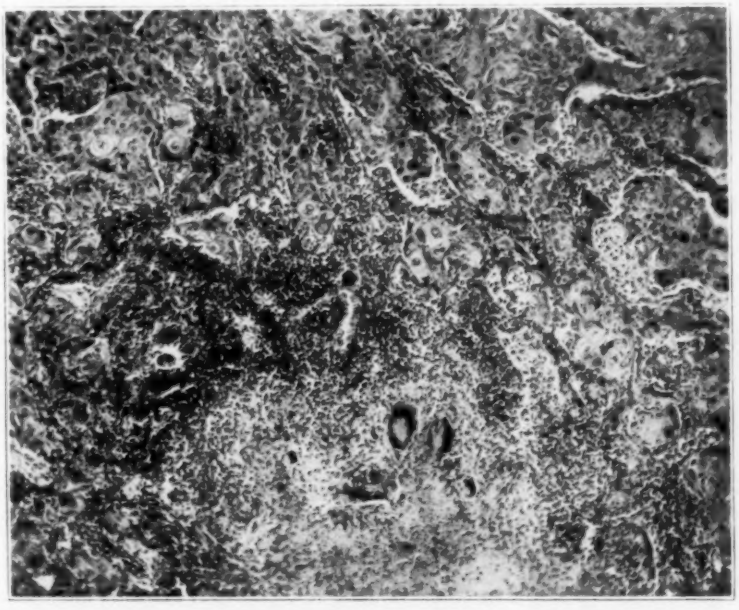

Fig. 2.—Microscopic section of lung showing invasion of tuberculous area by carcinomatous cells.

Microscopically, the tumor was composed of irregular papillary masses with a central cellular core. The cells covering these papillae were irregular in shape and size but were clear and deeply stained and showed a clear vesicular nucleus. The diagnosis was primary adenocarcinoma of the lung.

Comment.—The whole appearance of the patient was that of a person with gross cerebral disease, and the physical signs of pulmonary involvement were almost negligible. Unfortunately, no roentgenologic examination was made of the lungs, although it might have been just as inconclusive as the physical examination of the chest. Since this case appeared, however, a roentgenologic examination of the chest has been made a routine in cases of suspected tumor of the brain. This procedure has since possibly saved us from similar errors.

The fluctuation of signs and symptoms, as well as the frequent absence of signs of increased intracranial pressure in secondary carcinoma of the brain, has been noted before. Heinemann [5] and Maas [6] have thought that it is due to the gross destruction of tissue by the carcinoma cells without compression of the brain substance as a whole, the carcinoma merely substituting for the tissue destroyed.

The mental state of patients with cerebral carcinoma has been described by Morse,[7] Binswanger,[8] Pachantoni, Barrett [9] and Siefert.[10] The latter has described a clinical picture that he considers peculiar to this condition. The points emphasized are: (1) the advanced age of the patient; (2) the extraordinarily rapid failure of health; (3) the increased apathy and difficulty of thought; (4) peculiar alternation of delirium and comparative clearness; (5) peculiar disturbance of speech resembling that in general paralysis; (6) infrequency of choked disk; (7) pain in the neck, root symptoms and vesical disturbance; (8) weakness of the extremities and muscles supplied by the seventh nerve, especially on the right, and (9) changes in gait and tendon reflexes. The diseases most frequently mistaken for cerebral carcinomatosis are primary tumor of the brain, general paralysis and chronic alcoholic intoxication. Confusional states are common, and the patient may be maniacal.

Case 2.—A woman, aged 50, was brought to the clinic, Dec. 7, 1924, because of headaches and general weakness. Twelve months before, she had suffered

5. Heinemann, Jakob: Ueber die Metastasierung maligner Tumoren ins Zentralnervensystem, Virchows Arch. f. path. Anat. **205**:418, 1911.

6. Maas: Arch. f. Psychiat. **51**:359, 1913.

7. Morse, M. E.: Two Cases Illustrating the Pathological and Psychiatric Aspects of Carcinomatous Metastases in the Central Nervous System, J. Nerv. & Ment. Dis. **58**:408, 1923.

8. Bingswanger, Kurt: Atypische symptomatische Psychose bei allgemeiner und Gehirnkarzinomatose, Deutsche Ztschr. f. Nervenh. **78-79**:423, 1921.

9. Barrett, A. M.: Tr. Clin. Soc. U. Michigan **7**:57, 1915-1916.

10. Siefert: Ueber die multiple Karzinomatose des Zentralnervensystems, München. med. Wchnschr. **49**:826, 1902.

from a severe general headache that had become constant. It radiated into the neck and lower jaw. She steadily lost weight and strength and a month before, double vision and drooping of the left upper eyelid were manifested. About the same time, vomiting occurred every morning before breakfast. The patient had become somewhat dull and apathetic and was confined to bed on account of general weakness.

She was emaciated and cachectic, with a bandage over the left eye to prevent double vision. She was mentally clouded and initiated nothing. When asked, she complained of headache. There was a marked dulness over the left apex, with retraction of the chest wall and restriction of movement. A few râles could be heard in the left supraclavicular fossa and over the apex of the lung posteriorly. There was also edema above the clavicle and a boggy feel to the supraclavicular fossa. A small, hard lymph node was felt just above the middle third of the left clavicle. The left external rectus muscle was paralyzed, and the left upper eyelid showed slight ptosis. The optic disks were normal. The report of the roentgenologic examination of the chest was "diffuse infiltration of both lungs, probably miliary tuberculosis." The Wassermann test on the blood on two occasions was reported as strongly positive. The spinal fluid showed a negative Wassermann test, a positive Nonne test, eleven small lymphocytes; the fluid was straw-colored and under increased pressure. The temperature remained normal, and the leukocytes averaged 6,800. The pulse rate fluctuated between 120 and 70.

Course.—The patient was put on antisyphilitic treatment but failed steadily in strength. Toward the end the neck became rigid; Kernig's sign appeared and the patient became increasingly drowsy, dull and finally stuporous. Respiration became of the Cheyne-Stokes type, and death occurred, May 5, 1925.

Necropsy.—The diagnosis at necropsy was: (1) primary carcinoma of the left lung with metastasis; (2) old healed apical tuberculosis and pleuritis, and (3) internal hydrocephalus. Grossly, there was little to explain all the clinical manifestations. The left pleural cavity, except for its upper portion, was almost obliterated by many fibrous adhesions, and it contained 300 cc. of fluid. The left lung was contracted, collapsed and fibrous, and contained a gray, solid area at the apex. Both lungs contained many small firm discrete nodules varying from 1 to 5 mm. in diameter. Crepitation was present in the right lung. Both lower lobes were edematous and congested. There was an enlarged lymph node in the supraclavicular space which on dissection proved to be connected to the first rib and apex of the pleura by bands of fibrous tissue.

Gross examination of the meninges and brain revealed surprisingly few abnormalities. The pia-arachnoid and dura seemed normal on all surfaces except in a small area around the pineal gland where the pia was thickened. In the brain, seen with a hand lens, were a few tiny nodules under the pia and in the cortex of the hemispheres and cerebellum. In the left lobe of the cerebellum, just under the pia, there was one nodule about 1 or 2 mm. in diameter that might easily have escaped observation. This was the only thing observed grossly that could have been called a metastatic tumor in the cranial contents. Internal hydrocephalus of both lateral and third ventricles was fairly well marked, but was absent in the fourth.

Microscopic sections taken from the upper lobe of the left lung near the apex included a large portion of tumor tissue. In one section there was a mass of irregular, ill-differentiated polymorphic cells jammed together with

thin septums between cell columns. The cells were clear and well stained and had pale vesicular nuclei. Mitotic figures were numerous. There were bands of dense, well organized, connective tissue, infiltrated by the carcinomatous cells. At the lower edge of the section, where some lung tissue could be seen, there were well-formed tubercles with a caseating center, giant cells and zones of epithelioid cells surrounded by several layers of lymphocytes. Chains and columns of carcinomatous cells were seen invading the lymphocytic zones of several tubercles, and one was completely surrounded by a mass of carcinoma. The metastatic nodules showed the same kind of carcinomatous cell, but there was an attempt at formation of acini, which were irregularly shaped and filled with mucin. Sections taken from the brain showed a thickened pia-arachnoid infiltrated with carcinomatous cells. There were no vascular changes in the pia or signs of inflammatory reaction. The carcinomatous cells were of the same type as had been found in the lung, and they had infiltrated the septal prolongations of the pia and the perivascular spaces of the vessels springing therefrom. Carcinomatous cells could be seen passing with the blood vessels of the pia into the brain and forming nodules in the cortex. One relatively large nodule in the cerebellar cortex was composed of large, pale, epithelial cells arranged to form oval, round and flattened acini. There were papillary projections of the cells lining the acini into its lumen. The dura mater was not examined. The diagnosis of the tumor and the metastatic lesions was adenocarcinoma, primary in the lung, and probably arising from the bronchial epithelium or mucous glands.

Comment.—The difficulties in diagnosis were extreme. The presence of old apical tuberculosis and the signs of meningeal irritation were established satisfactorily. The positive Wassermann test brought in another complication, although the general appearance of the patient suggested diffuse carcinomatosis. The natural desire was to explain all of the symptoms on the basis of one disease, and cancer, tuberculosis or syphilis seemed equally inviting; but no one disease alone could explain the picture. Actually it was proved by necropsy that at least two of these conditions were present at the same time. Toward the end, pulmonary tuberculosis with miliary dissemination and secondary tuberculous meningitis seemed the most feasible diagnosis, but the relatively slight fever, the spinal fluid observations and the general appearance of the patient did not support this diagnosis. The course of the disease and the severity of symptoms were not in favor of syphilis. According to Morse, when no primary tumor has been apparent, the diagnosis has most frequently been tuberculous meningitis. Actually the association of pulmonary tuberculosis and carcinoma is not uncommon. Several cases of Wolf, in which active miliary tuberculosis was associated with carcinoma, suggested that the malignant process may light up latent tuberculous foci. Carcinoma may arise in the wall of tuberculous cavities. In old sclerosis, atelectasis and in reparative lesions of pulmonary tuberculosis may be found alterations of bronchial and pulmonary epithelium marked by considerable cellular overgrowth.[1]

PATHOLOGY

The pathology of meningeal carcinomatosis has been carefully studied by numerous observers, and more recently Morse and Hassin have reviewed the literature and added cases of their own. The primary source may be lung, breast or gastro-intestinal tract. Morse has noted that carcinoma of the stomach has been considered a common source by earlier observers. It seems to be established that the cerebral meninges may be involved without gross involvement of the cerebral substance, and this is more usually the case. By contrast, in cases showing cerebral involvement, the meninges are seldom involved secondarily except when a metastatic tumor of the brain has involved the membranes directly by its growth and extension, and then only in the vicinity of the tumor. Hassin believes that the meninges are involved by spread through the perineural lymphatics along the cranial nerves, the source being the metastatic cells in the lymph spaces and nodes of the neck. In Morse's case the membranes of the cord were involved in nearly its whole extent, and he assumed a spread thereto along the perineural lymphatics of the intercostal nerves. The actual changes in the meninges are described by Hassin, who found very few signs of reaction and no vascular changes. The only pathologic elements besides the carcinomatous cells were gitter cells, macrophages and plasma cells. There was therefore little justification for the diagnosis of meningitis. The brain underneath showed no carcinomatous cells, except where they had infiltrated along the septal prolongations of the pia-arachnoid. Hassin found that the brain showed parenchymatous changes combined with proliferation of the capillaries of the glial nuclei and the formation of a glial reticulum, which he interpreted as evidence of an inflammatory reaction or encephalitis. The tumor cells spread over the surface of the pia-arachnoid and the root sheaths by direct extension, in much the same way as the carcinoma spreads over the surface of the pleura or over the mucous membrane of the bronchi. The brain is involved in cases of meningeal carcinomatosis only to a short distance under the surface by infiltration of the carcinoma cells along the perivascular spaces of vessels entering the cortex from the meninges, both on the surface of the brain and in the prolongation of the pia into the cerebral and cerebellar sulci.

According to Morse, the symptoms most often reported in these cases of meningeal carcinomatosis have been "headache, vomiting, pain and stiffness of the neck and psychic disturbances of various kinds followed by a lapse into stupor. In some instances these symptoms developed rapidly at the onset of illness, in others they closed the scene after a variety of earlier manifestations." In the case just described there was only a mild apathy and dulness at the beginning which gradually progressed into drowsiness, stupor and finally coma. The

mental clouding and gradual decline with signs of meningeal irritation were the outstanding features of the case.

CASE 3.—A man, aged 53, came to the clinic, Dec. 11, 1924, because of pain in the left side of the chest. Eighteen months before, he had noticed a hypersensitive area over the skin of the chest on the left side, just above the nipple. This became painful, and the pain spread around the chest in a band 5 cm. wide, from the sternum to the vertebral spine, following closely the second and third segmental cutaneous areas or corresponding intercostal spaces. Five months later, the area spread to include the left upper extremity as far as the elbow on the medial surface. Occasionally the patient suffered from darting, tingling pains radiating down the arm to the hand. Light touch aggravated the pain so much that he preferred to discard his coat and vest when not in public and relieve the painful area of all pressure. Heavy pressure seemed to relieve the pain, which at times had a burning, stinging quality. For three months the pain had been constant day and night, disturbing sleep, and the patient preferred to walk around when it was severe, which was chiefly at night, so that he had lost considerable sleep. There were no other symptoms on the occasion of the first visit.

The patient was tall and gaunt, looking older than his years, and obviously was in severe pain. He paraded his room almost constantly with a loose shirt on, and occasionally with the whole left upper extremity, shoulder and left side of the thorax bare. He seemed in fair general condition and denied having lost weight. At the level of the root of the lung posteriorly, a few crackling râles could be heard and the percussion note was impaired over the apex of the left lung. No other signs of pulmonary disease were elicited at that time. He was exquisitely sensitive to light touch over the left second and third intercostal spaces, shrinking abruptly away from the fingers of the examiner. Roentgenologic examination of the chest showed changes diagnosed as "healed tuberculosis of upper lobe of left lung with upper mediastinal thickening." Repeated roentgenologic studies of the spine gave no result.

A diagnosis was made of root pain, with indeterminate causation, but on account of the severe pain symptomatic relief was deemed advisable. December 30, laminectomy was performed, and the first, second and third dorsal nerve roots were severed on the left, inside the dura. Nothing abnormal was encountered during this procedure. The spinal canal was free, and the spinal cord was normal. The vertebrae in that area seemed thicker and more vascular than normal, but microscopic sections of decalcified bone showed nothing unusual. The patient was immediately relieved of pain, made a speedy convalescence and returned home.

Four months later, April 23, 1925, he returned because of recurrence of pain, this time down the left arm as before but lower down on the thorax in the fourth and fifth interspaces. He had also had a severe attack of pain in the right lumbar region that radiated into the abdomen downward toward the pubes. This had lasted only a few hours. Pyrexia had developed, and the patient had noticed a swelling over the left scapula and upper part of the left trapezius muscle posteriorly.

The swelling was readily seen on examination and grew progressively larger up to the time of death. It was hard and firm and extended anteriorly into the supraclavicular space. The margins were indistinct, and percussion over it gave a dull note as found at the first examination. Breath sounds were diminished over the tumor, but no adventitial sounds were heard. Pyrexia

ranged between 102 and 103 F., and the leukocyte count varied between 15,000 and 17,000. The patient was anemic and much weaker than in December. Roentgenologic examination showed no change in the condition at the left apex, but "thickened pleura at the right base" was reported. A mass was felt in the pelvis by rectal examination, apparently larger on the right side. The only neurologic observations were those consequent to section of the nerve roots; that is, a segmental area of anesthesia occupying the second and third dorsal segmental areas.

The patient steadily declined during the two months he was under observation. The mass over the scapula and shoulder increased in size, and aspiration of it with a needle produced only a few cubic centimeters of bloody fluid. Toward the end, the left hand became edematous. A provisional diagnosis of sarcoma of the scapula with metastasis was made. Death from exhaustion and cachexia occurred, June 15, 1925.

Necropsy was limited to the tumor and the structures near it. Accordingly, the skin was reflected over the mass and the scapula disarticulated. Three or four ribs were removed, and the pleural cavity was entered in this way. The spinal canal was also opened. The pathologic changes were carcinoma of the left lung with invasion of the posterior wall of the thorax and the adjacent vertebrae, and extension bronchopneumonia. The tumor filled the subscapular fossa and was 5 cm. in thickness, soft yellowish white and gelatinous. The ribs, intercostal spaces, muscles and nerves were involved, and the origin of the tumor seemed to be from the apex of the lung posteriorly. The tumor had infiltrated along the intercostal nerves into the intervertebral foramina, penetrating to the dura which was apparently not involved. The spinal cord was intact. Both lungs were dark red, congested and without crepitations. On section, pus could be squeezed from the bronchi. The pleural surfaces were not involved.

Microscopic examination of the lungs showed, apart from the tumor, distinct evidence of bronchopneumonia with small abscesses. The lung tumor was composed of a mass of poorly differentiated, clear, polymorphic cells containing large clear nuclei. Many mitotic figures were present, and some areas showed acini. Sections from the pleura showed the same type of cell but more differentiation. There were numerous glandlike spaces lined by epithelial cells. In places the cells were infiltrating the pleural tissue in solid clumps of polyhedral cells. Sections taken from the first dorsal nerve where it was surrounded by tumor showed invasion of the nerve bundles by the tumor. There were carcinomatous cells even in between the nerve fibers of one individual fasciculus. The diagnosis was primary adenocarcinoma of the lung arising from the bronchial mucous glands.

Comment.—The sole complaint for more than eighteen months was that of root pain. Symptoms of pulmonary disease were in abeyance, and the physical signs at first were slight and could have been explained by old healed tuberculosis at the apex. The burning character of the pain and the extreme sensitiveness of the skin in the area involved suggested an involvement of the spinal ganglia or the posterior roots. The carcinoma in the lung had spread posteriorly and medially, and the phenomena of nerve irritation were the earliest signs. It is unfortunate that opportunity was not given for a more complete necropsy to

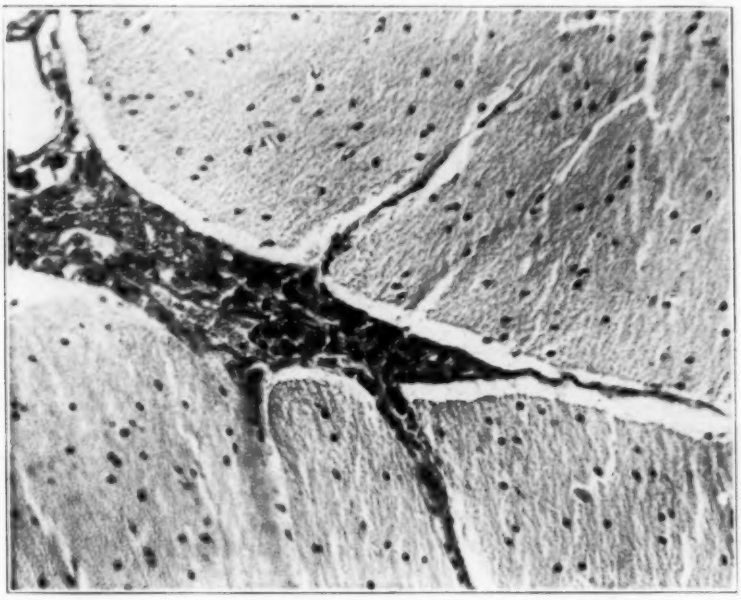

Fig. 3.—Meningeal carcinomatosis with extension along pial septums, and perivascular infiltration of vessels leading into the cortex of the cerebellum.

Fig. 4.—Infiltration of the nerve bundles of the second intercostal nerve by primary carcinoma of the lung.

rule out, beyond possibility of doubt, primary carcinoma elsewhere. The case cannot be said, therefore, to be complete. The fever and leukocytosis were misleading at first and suggested a deep-seated inflammatory process, but the negative results of aspiration and the general course of events were sufficient to suggest a carcinomatous process. Fever and leukocytosis in cases of pulmonary carcinoma are not rare. They are usually due to secondary infection of the ulcerating tumors in the bronchi or to bronchopneumonia or bronchiectasis. All five of Morse's patients had fever, and night sweats were present in one case. Fishberg and Steinbach [11] also consider fever, leukocytosis and night sweats common complications of pulmonary carcinoma. The temperature may have a diurnal variation rising in the afternoon to as high as 103 F.; its course may be continuous, remittent or intermittent.

CASE 4.—A man, aged 40, came to the clinic, May 8, 1925, complaining of pain in the back. Eight months before, following the lifting of a railroad hand-car, he experienced a sudden severe pain in his dorsal spine that radiated around in the seventh intercostal space to reach the xiphoid process in front. He was forced to go to bed, but within two days was much improved although some slight pain continued. Three months later, again following sudden exertion, the pain increased in severity and commenced on the right side, also at the same level. Movement of the back made the pain much worse, and he was relieved by lying flat on his back on a hard surface. He had lost 18 pounds (8.2 Kg.) in six months. There had not been any complaint directly referable to the respiratory system.

The patient was well developed, and showed signs of loss of weight. He held his back extremely rigid, and there was obvious muscle spasm in the erector spinae. There was extreme tenderness to percussion over the seventh dorsal vertebra, and roentgenologic examination of this region showed destruction of the body of the vertebra. Exaggerated breath sounds were heard over both lung apexes, but nothing else of importance was found. Roentgenologic examination of the chest was reported as "interlobar pleurisy at the third rib; possibly old inflammatory lesion; may be a small area of encapsulated pleura. The area on the left side at the fifth rib may be localized area of bronchiectasis." A diagnosis of Pott's disease was made, and the patient returned home.

On his return three months later, July 8, he complained of increasing pain in the same area as before, and also of pain in the left sacro-iliac joint. While under observation he suffered from severe right-sided headaches and vomiting, and became confused mentally at intervals. On one occasion he arose at night and, losing his way in the ward, urinated on the floor and had to be led back to bed. Next morning he was as bright mentally as ever. This fluctuating mental confusion continued, and examination of the eyegrounds showed an acute papilledema of from 4 to 5 diopters with hemorrhages and exudates. The spinal fluid was yellow and showed no response to jugular compression, although its mean pressure was as high as 30 cc. of water. The Nonne test was positive; the cell count was within normal limits. A complete neurologic examination showed no abnormality except a diminution in the right Achilles reflex. There was no paralysis, or sensory or trophic change. A diagnosis was

11. Fishberg, M., and Steinbach, M.: The Diagnosis of Intrathoracic Neoplasms, Med. Rec. **99**:513 (March) 1921.

made of "carcinoma with multiple metastasis to vertebra and brain, primary source unknown."

The pains in the hip and back improved under rest in bed and the wearing of a cast, but the headaches became increasingly severe with frequent vomiting and occasional spells of mental confusion. October 14, the patient was allowed to sit in a chair, but he suddenly fell over unconscious and died within a few minutes.

The diagnosis at necropsy was: (1) primary carcinoma of the left lung with metastasis to the right lung, regional lymph nodes, liver, suprarenals, prostate, brain, ribs, sixth and seventh dorsal and third, fourth and fifth lumbar vertebrae; (2) pulmonary embolism (source, left common iliac vein); (3) right parietal cyst obliterating the inferior horn of the lateral ventricle; (4) old healed tuberculous pleuritis and lymphadenitis.

Examination of the pleural cavities showed adhesions at the left pulmonary apex and at the base of the right lung. There were nodules on the left pleura posteriorly about the fifth and sixth ribs. The right lung on section showed many yellowish-white nodules scattered throughout its substance. The left lung contained a firm, hard, irregular mass in the lower lobe near the interlobar fissure. It seemed to radiate from the hilum along the bronchi, which were dilated in places. There was a large clot in the right pulmonary vessels. Metastatic nodules were found in all of the organs mentioned in the pathologic diagnosis. There was a large mass posterior and lateral to the left psoas muscle, extending from the last rib superiorly to the brim of the pelvis inferiorly. The tumor was firm and encapsulated, and was firmly attached to the transverse process of the third, fourth and fifth lumbar vertebrae. The psoas muscle, as well as the lumbar plexus of nerves, was pushed medially. The sixth and seventh dorsal and the third, fourth and fifth lumbar vertebrae were soft, crumbling and easily cut with a knife. The body of the seventh dorsal vertebra was crushed and flattened anteriorly. The spinal cord was not compressed. The meninges seemed normal grossly, and the brain was normal on the surface. On section of the brain, a large cavity, 8 cm. in diameter, filled with yellow blood-tinged fluid, was found in the right cerebral hemisphere at the beginning of the inferior horn of the lateral ventricle, into which it bulged without communicating with it. It occluded the posterior horn and prevented communication between the inferior horn and the anterior horn of the right lateral ventricle. It was separated from the ventricular cavity mainly by a layer of ependyma and the cyst wall. One small metastatic nodule, 1 cm. in diameter, was found in the left cortex over the left superior temporal gyrus.

Microscopic sections were taken from the left lung in the region of the primary growth and from the region invaded by metastatic nodules. Sections from the tumor area showed the aveoli filled with large irregularly shaped epithelial cells with large vesicular dark stained nuclei. The alveolar wall was preserved except for its lining. Mitotic figures were numerous in the tumor cells. The cells in general had no regular arrangement but were in masses conforming to the shape of the alveoli. Occasionally a few cells were grouped to form something suggesting an acinus. A small bronchus in the section was invaded by masses of carcinomatous cells, which infiltrated the walls and narrowed the lumen and at one point passed through the bronchial epithelium. The metastatic nodules in both lungs showed more differentiation, their cells being arranged into acinous or glandlike formations, irregularly shaped. The hilum nodes were almost completely destroyed by carcinoma cells replacing the lymphoid tissue. There were numerous carcinomatous cells in the

tumor removed from around the third, fourth and fifth lumbar vertebrae. They were the same type as seen in the lung. Their general tendency was to be arranged in acini, but the order was poor. Sections through the wall of the cyst in the brain showed carcinomatous cells composing the wall with degeneration in the center forming the cavity of the cyst. The diagnosis was primary adenocarcinoma of the lung arising from the bronchial mucous membrane.

Comment.—Apparently the initial symptoms of backache and root pains were caused by the collapse of the seventh dorsal vertebra weakened by a metastatic tumor. The mere effort of lifting a heavy weight was responsible for this, and the symptoms recurred later under similar circumstances. The resemblance to Pott's disease, at least superficially and at the beginning, was close, and yet the severity of the pain and the age of the patient should have made one suspicious. Later, the appearance of pain in the hip and cerebral symptoms with progressive failure were sufficient to suggest widespread malignant disease. As in the other cases, there were no pulmonary symptoms, and the roentgenologic examinations were inconclusive and misleading.

The course of the disease was fairly rapid, taking only twelve months from the appearance of the first symptom to death from pulmonary embolism. But for this more rapid course, this case is almost an exact counterpart of another which was seen here in 1920. Death occurred elsewhere and necropsy was performed by Barron, who included the case in his series of thirteen proved cases of carcinoma of the lung. In this case also a mistaken diagnosis of Pott's disease was made, and the course was of three years' duration with a sudden onset of backache and root pain while the patient was playing golf. Weakness of the legs appeared and was so increased by chiropractic treatment that he was unable to walk. There was a knuckle deformity in the region of the twelfth dorsal vertebra and local anesthesia as high as the eleventh dorsal segment. There were no symptoms of pulmonary involvement, and both physical and roentgenologic examinations of the lungs were negative. Large lymph nodes could be felt in the left inguinal region and could also be felt in the pelvis by rectal examination. At necropsy a primary carcinoma of the left lung was found in the lower lobe around the bronchi. Microscopically, the tumor was scirrhous carcinoma originating from the mucous glands of the bronchus.

GENERAL COMMENT

The four cases described belong to the clinical group already mentioned wherein the early and dominant symptoms are those of invasion of the nervous system, and the primary carcinoma remains latent and produces few or no signs. Malignant disease was diagnosed in three cases, but that it was primary in the lung was not considered. In only one case was there obvious clinical evidence of gross pulmonary disease;

in the other three, the signs of pulmonary invasion were scanty, indefinite and easily misinterpreted. In no instance did the patient complain of cough, dyspnea or pain associated with respiration, and in each case questions were directed as a routine to the status of the respiratory system. Fried [12] reported ten cases of pulmonary carcinoma with cerebral involvement in five. Four of these patients sought advice because of cerebral symptoms alone, and in three of these no diagnosis had been made prior to death. A diagnosis of alcoholic psychosis was made in the case of the fourth patient. Barron reported thirteen cases of carcinoma of the lung in which he had performed necropsy; a correct diagnosis had been made in only three instances. In the five cases reported by Moise,[13] a correct diagnosis was made in only two. On the other hand, Fishberg and Steinbach claimed to have failed only once to make a correct diagnosis in thirty-three cases during a period of five years.

SYMPTOMS

The chief complaint and earliest symptom in the four cases in the present series were referable to the nervous system: in two cases headache and vomiting, and in the other two root pains. In one case headache and vomiting appeared toward the end. In case 2, there was marked involvement of the meninges with carcinoma and only minute and scanty carcinomatous nodules in the cerebral and cerebellar cortex. In case 1 there were many gross metastatic nodules deep in the brain. The symptoms in the case of meningeal carcinomatosis were headaches and ocular paralysis in the beginning, and only shortly before death did rigidity of the neck, Kernig's sign and stupor appear. In the case of the patient with multiple cerebral metastasis, the signs of cerebral destruction appeared early and consisted of transient aphasia, monoplegia and mental deterioration. This might suggest a differential diagnosis between meningeal and cerebral carcinomatosis if they were present in the majority of cases, but the experience of others is not in accord with ours. The literature as reviewed by Moise led him to the conclusion that the clinical picture was so complicated and confused that the value of differential points in any particular case would be limited.

The root pains complained of by two of three patients were of the usual type associated with compression of a nerve root by carcinoma. In one (case 3), the pain was due to direct involvement of the second and third dorsal roots by the tumor, and in the other (case 4), to crumbling of the bodies of the vertebrae. Root pain high in the

12. Fried, B. M.: Primary Carcinoma of the Lungs, Arch. Int. Med. **35**:1 (Jan.) 1925.

13. Moise, T. S.: Primary Carcinoma of the Lungs, Arch. Int. Med. **28**: 733 (Dec.) 1921.

dorsal segmental zone should suggest direct compression of the intercostal nerves by a mediastinal tumor, aneurysm or carcinoma of the lung. The difficulty lies in demonstrating the particular cause of compression and consequent pain. If the signs of intrathoracic disease, such as cough, cyanosis of the face and hoarseness, are absent, it is difficult to exclude tumor arising inside the spinal canal or spinal dura. Destruction of a vertebra by metastasis may simulate Pott's disease, as in case 4; but the course is relatively short and the pain much more intense and constant. Tuberculosis is relatively a slower process than carcinoma, and the extreme pain on movement, percussion of the spine, coughing or even the shaking of the bed by the examiner, is more suggestive of malignant than of tuberculous disease of the spine. Signs of intracranial disease were added to the signs of vertebral destruction in case 4. These might have been mistaken for cerebral or meningeal tuberculosis but for the appearance of the spinal fluid and the relatively rapid progress of events.

The roentgenologic observations were just as difficult to interpret as the clinical signs and symptoms, and as liable to misinterpretation. In case 2, there was an area of tuberculosis in the apex of the left lung, and the metastatic nodules in the two lungs were falsely interpreted as miliary dissemination of the tuberculous lesion. In another instance (case 3), the tumor arose from the apex (which is not the usual origin of pulmonary carcinoma) and grew out through the thoracic wall under the scapula where its shadow was obscured by that bone. In case 4, the carcinoma was not well advanced and was mistaken for an area of bronchiectasis. As McMahon and Carman[14] have pointed out the areas of density in carcinoma of the lung, while in an early stage, may not be entirely typical; in such cases the roentgenologic diagnosis can only be tentative. In any individual case the diagnosis must be made by analysis of all data, whether clinical or laboratory, and irrespective of what system the disease happens to strike the hardest.

SUMMARY

In four cases of primary carcinoma of the lung with invasion of the brain, meninges, spine and nerve roots, the carcinoma gave few signs, and the involvement of the nervous system was the most striking feature in each case. The clinical and necropsy records in each case illustrate the great variability in the course and tendencies of the disease and the difficulties in diagnosis. The study demonstrates the necessity of a complete general physical examination in every case of disease of the central nervous system.

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14. McMahon, F. B., and Carman, R. D.: The Roentgenologic Diagnosis of Primary Carcinoma of the Lung, Am. J. M. Sc. **155**:34 (Jan.) 1918.

GENERAL PARALYSIS

THE HISTOPATHOLOGY OF THE BASAL GANGLIA, CORPUS CALLOSUM AND DENTATE NUCLEUS IN FOUR CASES *

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OMAHA

My object in this paper is to describe the pathologic changes found in the basal ganglia in ordinary cases of general paralysis; not only are the changes definite and characteristic but also some of the signs typical of the disease may be explained on the basis of this pathologic condition. Such symptoms are the masklike or slightly expressionless facies, the speech defect and the involuntary tremors or twitchings of the small muscles of the face, especially about the eyes and the mouth.

In reviewing the literature of general paralysis, I have been able to find no detailed pathologic examination of the basal ganglia. Mella and Katz,[1] in reporting a case of neurosyphilis with a parkinsonian syndrome, make this statement: "Inquiry at various pathologic laboratories has shown that the neuropathologic investigation of syphilis has usually been limited to a study of the cortex, medulla and spinal cord, neglecting the basal ganglia." Also, "The basal ganglia of a series of syphilitic brains should be studied to determine the frequency of pallidal cell degeneration in syphilitic patients. . . ."

Jacob,[2] in a discussion of the changes in the striatum in general paralysis with choreiform movements, says: "We may also postulate certain components of paralytic speech and locomotor disturbance to the usual alterations in the striatum."

Dercum [3] says: "Our increasing knowledge of the striatum justifies, I believe, definite conclusions as to its functions, and among these to the rôle it may play in speech disturbances." That speech disturbance and involuntary tremors of the muscles of the face may be due to degeneration of the nerve cells of the corpus striatum is seen as part of some

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* From the Laboratories of Neuropathology and the wards of the Philadelphia General Hospital.

* Thesis for Degree of Master of Medical Science in Neuropsychiatry, University of Pennsylvania Graduate School of Medicine.

1. Mella, Hugo; and Katz, S. E.: Neurosyphilis as an Etiologic Factor in the Parkinsonian Syndrome, J. Nerv. & Ment. Dis. **59**:226 (March) 1924.

2. Jacob, A.: Anatomy, Clinical Syndromes and Physiology of the Extrapyramidal System, Arch. Neurol & Psychiat. **13**:596 (May) 1925.

3. Dercum, F. X.: Lesion of the Left Lenticular Nucleus and Adjacent Structure, Arch. Neurol. & Psychiat. **12**:601 (Dec.) 1924.

now well-known nonspecific clinical and pathologic syndromes of the basal ganglia.

Hunt [4] reports four cases of paralysis agitans (two of the juvenile type), all of which showed clinically a disturbance in speech, one a tremor of the face, tongue, eyes and eyelids, and one a tremor of the lips and eyelids. The histologic examination in one of these cases showed definite pathologic change in the cells of the corpus striatum. Hadfield [5] reports the case of a girl, aged 12½, with hepatolenticular degeneration, who one year before death developed difficulty in speaking, rigidity of musculature, a twist of the face and eyes and a change in facial expression. Histologic examination revealed pathologic change of the cells of the corpus striatum.

Wilson [6] in the original monograph on the disease which now bears his name, reported, among others, three cases of progressive lenticular degeneration in all of which a bilateral degeneration of the lenticular nucleus was found at necropsy. All three patients showed as the earliest symptom a disturbance in speech, a tremor of the extremities and later, a change in facial expression; ultimately all the patients developed complete anarthria, marked tremors of the extremities and contracture of the skeletal muscles. Stern [7] in reporting on the pathologic changes of chronic progressive or Huntington's chorea, cites three cases all of which, in addition to the choreiform movements of the face, arm and leg, presented a choreic defect in speech. Pathologic changes were found in the basal ganglia, especially the caudate nucleus and putamen, as well as the typical and definite changes in the deeper layers of the cortex. Campbell and Morse [8] report a case showing the pathologic changes of Westphal-Strümpell pseudosclerosis in which speech was defective, "slow and slurring," and also showing marked coarse tremors of the head, tongue, arms and legs.

All tissue in the cases here reported was fixed in formaldehyde; paraffin, celloidin and frozen sections were studied. Hematoxylin-eosin, Nissl, Bielschowsky, phosphotungstic acid, Marchi and Weigert stains were used. In case 3, tissue was stained by the Jahnel and Levaditi methods for *Spirochaeta pallida*. Some suggestive but no typical spirochetes were found.

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4. Hunt, J. R.: Progressive Atrophy of the Globus Pallidus, Brain **40**:58 (May) 1917.

5. Hadfield, Godfrey: On Hepatolenticular Degeneration with the Account of a Case and the Pathologic Findings, Brain **46**:147 (July) 1923.

6. Wilson, S. A. K.: Progressive Lenticular Degeneration, Brain **34**:296 (March) 1911-1912.

7. Stern, Felix: Beiträge zur Pathologie und Pathogenese der Chorea chronica progressiva, Arch. f. Psychiat. **63**:37, 1921.

8. Campbell, C. M., and Morse, M. E.: A Case of Westphal-Strümpell Pseudosclerosis Following Erysipelas, with Discussion of Allied Conditions, J. Neurol. & Psychopath. **5**:28 (May) 1924.

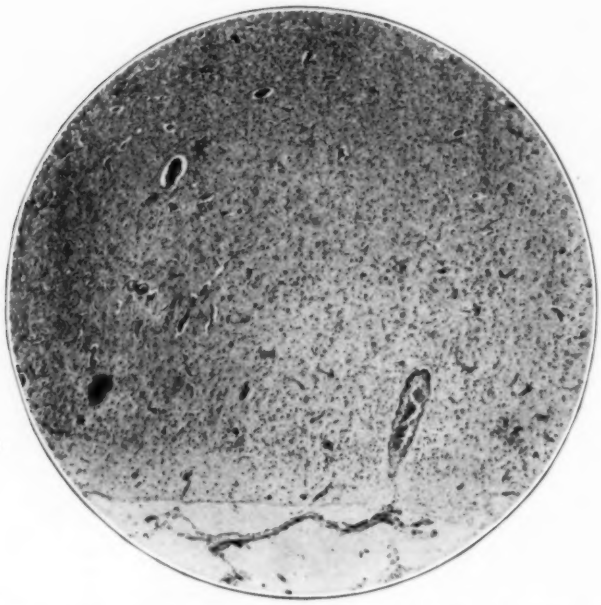

Fig. 1 (case 1).—Frontal cortex, showing marked disarrangement of the cellular architecture; perivascular infiltration.

Fig. 2 (case 1).—Optic thalamus, showing degeneration of large sensory ganglion cells; one cell dropped out; satellite cells slightly increased.

REPORT OF CASES

CASE 1 (Service of Dr. Gilpin).—*Basal ganglia symptoms: paretic facies, speech defect, tremor of muscles about face.*

History.—J. H., a white man, aged 41, had had mental symptoms for two years; he was irritable, impulsive, threatened and attacked his family, was unable to work and presented marked mental deterioration.

Neurologic and Laboratory Examination.—The pupils were irregular and unequal with poor reaction to light. The patient had a "paretic look." The facies was stupid; speech was thick; there was marked tremor of the muscles about the mouth. Coarse tremor was present in the outstretched fingers; the deep reflexes were hyperactive. The blood Wassermann reaction (cholesterinized antigen) was 3 plus. Examination of the spinal fluid showed: cells, 40; globulin, heavy trace; Wassermann reaction with both antigens, 4 plus; the colloidal gold curve was 4555335210.

CASE 2 (Service of Dr. Bochroch).—*Basal ganglia symptoms: speech defect, tremors of eyelids, lips and facial muscles.*

History.—A. R., a negro woman, aged 36, had had gradually failing vision for eighteen months, had been acting strangely for three months, and had had incontinence of urine and feces for two weeks. Mentally she showed an advanced stage of dementia.

Neurologic and Laboratory Examination.—The patient was unsteady in walking; the pupils were irregular, and did not react to light. There were marked tremors of the eyelids, facial muscles, lips and fingers. All deep reflexes were exaggerated. The blood Wassermann reaction (cholesterinized antigen) was 3 plus. The spinal fluid Wassermann reaction with both antigens was 4 plus.

CASE 3 (Service of Dr. McCarthy).—*Main symptoms: convulsions and mental deterioration.*

History.—H. D., a negro man, aged 40, had deteriorated to an imbecile condition in four years; previously he had been strong and healthy; he had had occasional convulsions with unconsciousness, and died from a fall in one of them.

Neurologic Examination.—The pupils were unequal, with slight reaction to light; the left eye turned outward. Tests for the deep reflexes gave no response. The spinal fluid showed increased globulin.

CASE 4 (Service of Dr. Rhein).—*Basal ganglia symptoms: speech defect, marked tremors of muscles of face.*

History.—E. F., a white man, aged 28, had showed marked mental symptoms and had been unable to work for more than a year; he acted strangely, laughed without cause and talked wildly to himself.

Neurologic and Laboratory Examination.—The pupils were unequal, irregular and reacted sluggishly to light; the tongue showed a very fine tremor; twitchings were noted in the muscles of the face; there was marked speech defect "indicative of paresis." The deep reflexes were exaggerated but equal. The blood Wassermann reaction with both antigens was 4 plus. The spinal fluid showed: cells, 8 per cubic millimeter; globulin, heavy trace; Wassermann reaction with both antigens, 4 plus; colloidal gold curce, 5555555421.

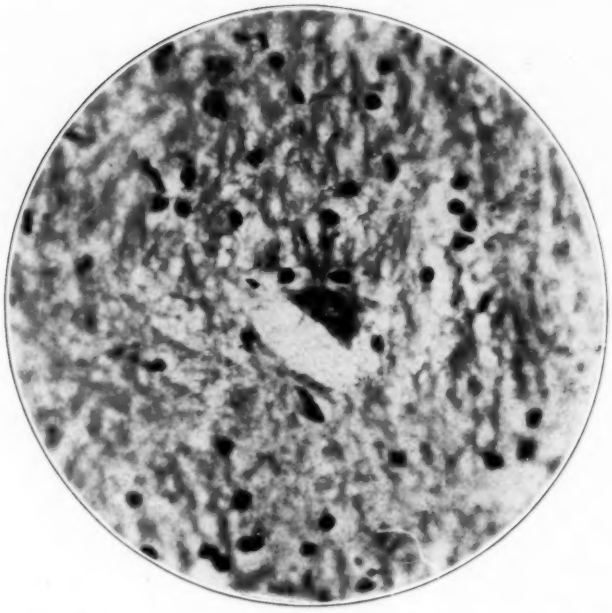

Fig. 3 (case 2).—Globus pallidus, showing satellitosis of large ganglion cell; early acute degenerative changes; cytoplasm granular.

Fig. 4 (case 2).—Putamen, showing degeneration of large and small ganglion cells; moderate satellitosis; slight collection of round cells in perivascular lymph space.

PATHOLOGIC OBSERVATIONS

CASE 1.—*Macroscopic Examination.*—There was marked atrophy of the cerebral convolutions, with thickening, roughening and opacity of the pia-arachnoid. There was a moderate degree of arteriosclerosis. On cross-section, the basal ganglia showed no abnormalities. The dura of the spinal cord was thickened.

Microscopic Examination.—Cortex: The pia-arachnoid showed a slight but definite infiltration with round cells, mainly lymphocytes and plasma cells. The cortical architecture was markedly disturbed by a focal loss of ganglion cells with an increase of glia arranged in an irregular manner. The vessels stood out prominently because of their thickened walls; in the zone of perivascular infiltration were lymphocytes and plasma cells. There were marked syphilitic changes in the intima. New blood vessel formation was pronounced. The anterior horn cells of the cord showed degenerative changes, especially chromatolysis and atrophy.

Basal Ganglia; Globus Pallidus: The blood vessels showed marked perivascular infiltration with round cells, most of which were small lymphocytes, but there were many plasma cells and large lymphocytes. There was marked proliferation of the endothelial cells of the vessels; budding and new blood vessel formation was seen and was fairly extensive, but not so marked as in the cortex.

The large motor ganglion cells appeared fewer than normal and many, though not all, of the remaining cells showed definite evidence of acute and chronic degeneration; some of the affected cells were shrunken, narrow and elongated; others showed axonal chromatolysis and moderate and severe neuronophagic changes. Some cells contained an excessive amount of lipoid material, which caused in many a definite bulging of the cell wall. Many cells were larger than normal and much fragmented; of these, some still contained particles of nuclei; in others the nucleus had disappeared. Many rod cells existed both near the vessels and scattered about the motor cells, and numerous proliferating glia cells, of varying size and shape, were present. Glia fibrils were increased.

Putamen: The blood vessel changes were definite and about the same as those described in the globus pallidus. Rod cells and proliferating glia cells were fairly numerous. The cellular architecture showed marked departure from the normal arrangement. Numerous axonal chromatolytic changes of both the large and the small cells were seen; satellitosis of from three to four neuroglia cells and neuronophagia of from four to five glia cells were frequently observed, the small cells appearing to suffer more neuronophagic changes than the large ones.

There were many areas in which a motor cell appeared to have dropped out, and the space was partly filled with faintly staining glia fibers. Severe phagocytosis of a few cells was present. In many of the small ganglion cells, the nucleus appeared large for the cell and usually contained heavy granules; the cytoplasm of such cells contained no granules. Many of the large ganglion cells were irregularly shaped, the nucleus being eccentric. A few of the large cells showed much fragmentation or granulization of both the nucleus and the cytoplasm.

Caudate Nucleus: The vessels of the caudate nucleus, especially those near the ventricle, showed more extensive perivascular infiltration than did those of the globus pallidus or putamen; about some vessels the exudation was more in amount than about most of the vessels of the cortex. The large and small

motor ganglion cells showed greater changes near the ventricular surface than deeper in the structure. In the same area, budding and new blood vessel formation were more extensive, and there was also a greater disturbance of the cellular architecture. Satellitosis and neuronophagia of numerous ganglion cells were present. The large and small polygonal cells showed evidence of acute and chronic degeneration of the acute degeneration type; many contained excessive deposits of fat. Neuroglia fibers appeared increased.

Optic Thalamus: Perivascular infiltration was marked throughout, but more severe in the portion bordering on the ventricle; small lymphocytes and plasma cells predominated; proliferating glia cells were numerous; many small lymphocytes were present, scattered throughout the nucleus. Satellitosis and neuronophagia of from five to six glia cells was frequently seen. In a great number of the large sensory cells the nucleus was eccentric, and the limiting cell membrane was broken up, and in many places had almost entirely disappeared. Many of the cells showed vacuolization of the nucleus and cytoplasm. Some of the cells stained faintly, and in many of these the nucleus was pyknotic with the nucleolus not apparent, the membrane of the nucleus and cell having broken down. There was evidence of axonal chromatolysis in many cells, and frequently cells were seen containing excessive amounts of faintly staining lipoid material. Phagocytosis of the large sensory cells was occasionally found, mostly near the ventricular portion of the thalamus. Many cells were exceptionally large, their limiting membrane having broken down; the nucleus in some of these cells was still present; in others it had disappeared. In some cells the nucleus was excentric, in others partly extruded.

Nucleus of Luys: Slight, but definite perivascular infiltration was present, not all the vessels showing it. New blood vessel formation was present. Proliferating glia cells and an occasional rod cell were seen. Some polygonal motor cells showed evidence of acute degeneration, although the majority showed no pathologic change. Satellitosis was present with an occasional neuronophagia of from two to three glia cells. Some of the cells showed vacuolization of the nucleus; many of the diseased cells showed an excentric nucleus containing granules; the limiting cell membrane was broken down, and the cytoplasm also contained many granules. Many of the cells contained an excessive amount of lipoid material.

Substantia Nigra: Perivascular infiltration, moderate in amount, was present. Rod and proliferating glia cells were present. Some of the substantia nigra cells showed vacuolization of the nucleus and cytoplasm; many showed evidence of acute degeneration with swelling; others showed portions of the pigmentary substance gone; in many of these the nucleus was present; others showed extrusion of the nucleus and a breaking up and disappearance of the cellular substance. Satellitosis was present; also, an occasional neuronophagia of two or three cells was seen. In places only the remnants of substantia nigra cells remained.

Dentate Nucleus: Many of the blood vessels showed no evidence of perivascular infiltration or syphilitic changes; those involved showed, in the perivascular lymphatic space, a number of small lymphocytes and a few plasma cells and large lymphocytes. New blood vessel formation was scarce. A few rod cells and a fair number of proliferating glia cells were present. Some of the dentate cells showed evidence of acute degeneration; no chronic cell changes were present, and the large majority of cells showed no pathologic changes. Several of the cells appeared large and swollen; the cytoplasm contained granules, and in some cells the nucleus and limiting cell membrane

had broken down and partly disappeared. Many of the ballooned cells contained a large excess of fat in meshes. The nuclei in a fair number of cells were in the various stages of early degeneration.

Red Nucleus: Perivascular infiltration here was not so severe as in the striatum; new blood vessel formation was present, and many proliferating endothelial cells were seen. Rod cells and proliferating glia cells of varying size and shape were fairly constant. There was definite degeneration of both large and small ganglion cells; axonal chromatolysis, satellitosis, neuronophagia and phagocytosis of cells were seen. The degeneration of the cells in this area, however, was not as marked as in the striatum.

Corpus Callosum: Some but not all vessels showed slight round cell infiltration in the perivascular lymph spaces; these were mostly small lymphocytes. A few rod cells and proliferating glia cells were present. Increased blood vessel formation was not apparent.

Case 2.—*Macroscopic Examination.*—There was thickening of the dura. The pia-arachnoid was thickened, especially over the frontal and parietal lobes. There was marked convolutional atrophy with pronounced widening of the sulci; moderate arteriosclerosis of the basal vessels was present. Cross section showed no gross pathologic change of the basal ganglia, but there was slight dilatation of the ventricles with definite ependymitis of their walls.

Microscopic Examination.—Cortex: The pia-arachnoid was thickened and markedly infiltrated with round cells, mainly lymphocytes and plasma cells. The cellular architecture of the cortex was markedly disturbed and showed evidence of disappearance of the ganglion cells. There was increase of glia cells with marked irregularity in their placing. The cortical vessels stood out prominently because of the "cuffing" with round cells. Plasma cells and new vessel formation were present. The cord showed fat accumulation in the cells with vascular thickening.

Caudate Nucleus: The blood vessels showed marked perivascular infiltration, and many of the vessels near the ventricle margin had a definite cuffing of round cells in which small lymphocytes and plasma cells predominated. Rod cells, proliferating glia and endothelial cells were abundant. Both large and small ganglion cells showed evidence of acute and chronic degeneration, the acute predominating. Neuronophagia of three and four glia cells was frequently seen. A large collection of lipoid material bulged from a side or end of some of the cells. Many of the cell walls were entirely broken down, and in the cytoplasm were several glia cells and some scattered, faintly staining granular substance; the whole cell was in a process of advanced disintegration. Increased glia fibers were present. All the pathologic changes were more severe near the ventricle than deeper in the structure.

Globus Pallidus: Perivascular infiltration of round cells was present but in most areas was less in amount than that found in the putamen; small lymphocytes and plasma cells predominated. Rod cells, new blood vessel formation and proliferating glia cells were constant. In some areas, mostly in the mesial segment, were spaces in which large pallidal cells appeared to have dropped out. Satellitosis and neuronophagia of three and four glia cells were present. Many of the cells contained no nucleus, and the cytoplasm stained faintly Both the large and small cells showed evidence of severe, acute and chronic degeneration. Increase in the neuroglia fibers was apparent.

Putamen: Changes similar to those described in the caudate nucleus were found in the putamen; however, the perivascular infiltration was not as great nor were the degenerated ganglion cells as numerous as in the caudate nucleus; when present they were quite as severe.

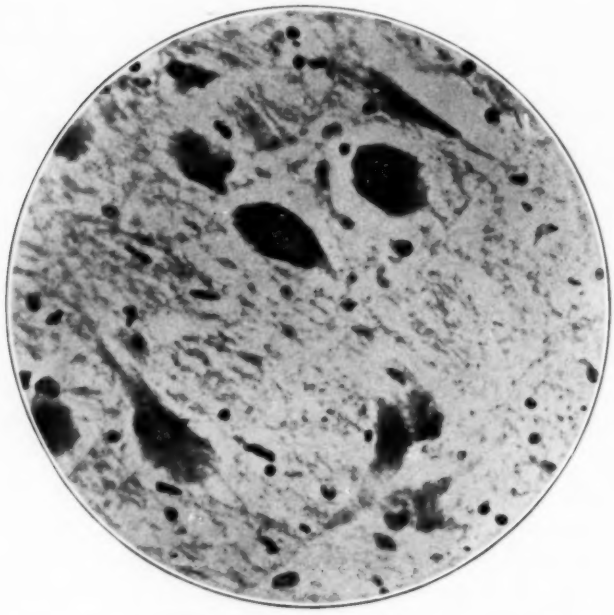

Fig. 5 (case 2).—Substantia nigra, showing degeneration of two cells; structure of others hidden by pigment.

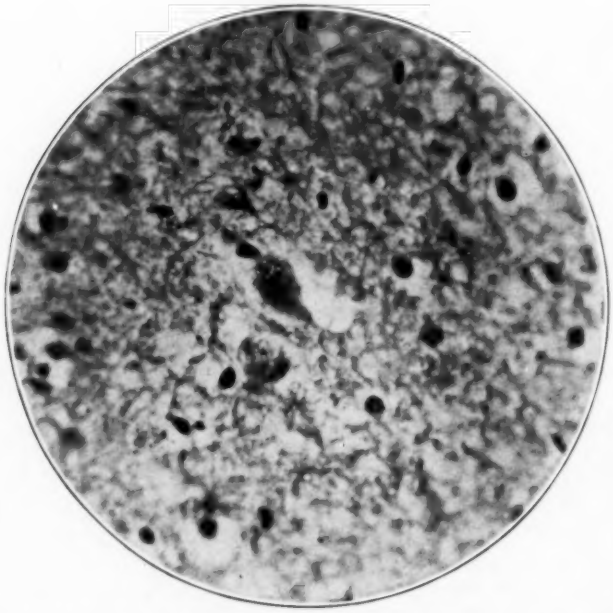

Fig. 6 (case 2).—Body of Luys, showing acute degeneration of ganglion cells.

Optic Thalamus: Perivascular infiltration of the vessels occurred throughout the entire structure; the small lymphocytes were in excess, but plasma cells and large lymphocytes were numerous. Large proliferating endothelial cells were present in the vessel walls. Rod cells, new blood vessel formation and proliferating glia cells were numerous. Degeneration of the thalamic sensory cells was seen throughout, but was most extensive near the ventricular border. Satellitosis of six and seven cells and neuronophagia of three and four glia cells were frequently seen. The nuclei of many cells were eccentric. Vacuolization of both nucleus and cytoplasm occurred, and excessive amounts of lipoid substance were seen in some cells. In some cells the nucleus was large and contained empty spaces; in others the nucleus could not be made out; the cell membrane was broken down and what remained of the cells was an

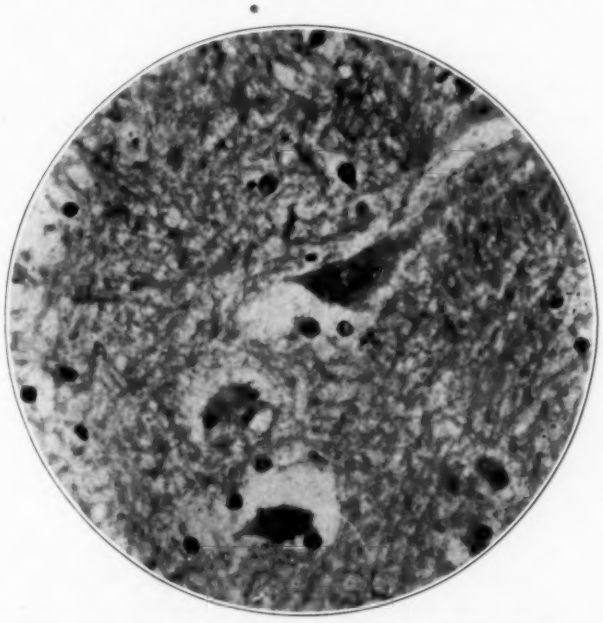

Fig. 7 (case 2).—Red nucleus, showing marked degeneration of two small ganglion cells; slight degeneration of large ganglion cells.

irregular collection of fine granular, faintly staining substance, in one portion of which was usually seen a fair sized deposit of lipoid substance.

Nucleus Ruber: There was a moderate degree of perivascular infiltration; not all vessels showed infiltration; some had only one or two small lymphocytes in the perivascular lymph space. Proliferation of glia cells and fibers was moderate in amount. Satellitosis of four and five glia cells, neuronophagia and degeneration of both the large and the small motor cells were present, but were not as extensive as in the striatum. The nucleus was eccentric or absent from many cells; some showed marked fragmentation of both nucleus and cytoplasm; others bulged at the side or end with heavy deposits of lipoid material.

Substantia Nigra: The vascular changes were about the the same as those found in the nucleus ruber. The nigra cells showed various stages of degeneration; some were swollen, others pale and shrunken. The nucleus was extruded

from some; in others it was absent; some showed disappearance of part of the pigment substance. Only fragments of both nucleus and cytoplasm of many cells remained. Rod and proliferating glia cells were present.

Nucleus of Luys: Perivascular infiltration was present but not as extensive as in the red nucleus; many of the cells here showed acute and chronic degenerative changes, some quite severe. Rod cells and proliferating glia cells were present, and the glia fibers appeared increased.

Dentate Nucleus: Slight perivascular infiltration was found; not all the vessels, however, showed it. There was proliferation of the endothelial cells, and new vessel formation was seen. Rod cells and proliferating glia cells were present but not numerous. Many of the large dentate ganglion cells showed evidence of degeneration; some were without nuclei; others showed the nuclei greatly distended and partly filled with heavy and light staining substance; slight vacuolization was also present in some of these cells. Satellitosis and neuronophagia were occasionally seen.

Corpus Callosum: Some of the vessels, but not all, showed a few round cells in the perivascular space; no changes in the vessel walls were noted; budding and new blood vessel formation were not observed. Rod cells and proliferating neuroglia cells were occasionally seen.

Case 3.—*Macroscopic Examination.*—The brain was small and the pia-arachnoid thickened, most markedly over the frontal and parietal lobes. There was marked convolutional atrophy, especially severe over the frontal lobes. The basal vessels were not particularly thickened. Cross-sections showed no gross abnormalities of the basal ganglia, but fairly marked ventricular dilatation. The dura of the cord was thickened.

Microscopic Examination.—Cortex: The cortex, especially in the precentral area, presented marked disarrangements of the cell layers, the chief dendrites of the neurons being misdirected and the general architecture of the cortex much disturbed. There was new formation of blood vessels. There was marked perivascular infiltration with small round cells. There was a general increase of irregularly scattered neuroglia, and empty spaces were seen which may have been occupied by nerve cells. The pyramidal cells were altered and showed evidence of chronic cell degeneration. The nuclei were shrunken and eccentrically placed; vacuolization and instances of neuronophagia were seen.

Caudate Nucleus and Putamen: There was marked perivascular infiltration in the vessels of the caudate nucelus, and in the tail and near the ventricular border it was almost as severe as that found in the cortex. The perivascular infiltration of the putamen was not as marked as that of the caudate. Small lymphocytes and plasma cells predominated. There was proliferation of the endothelial cells of the vessels. Rod cells and proliferating glia cells were numerous. The large and small motor cells of the caudate nucleus and putamen showed evidence of acute and chronic degeneration. Satellitosis and neuronophagia of three and four glia cells were frequently seen. In places large and small polygonal cells seemed to have dropped out. Severe phagocytosis was occasionally encountered. Phosphotungstic acid stain showed a slight general increase in neuroglia fibers. Pathologic changes of all kinds were more severe in the tissue near the ventricles than away from it.

Globus Pallidus: The vascular changes were not so marked as those in the putamen; some hyalinization of the vessels was present. New blood vessel formation was seen. Rod and proliferating glia cells were fairly numerous.

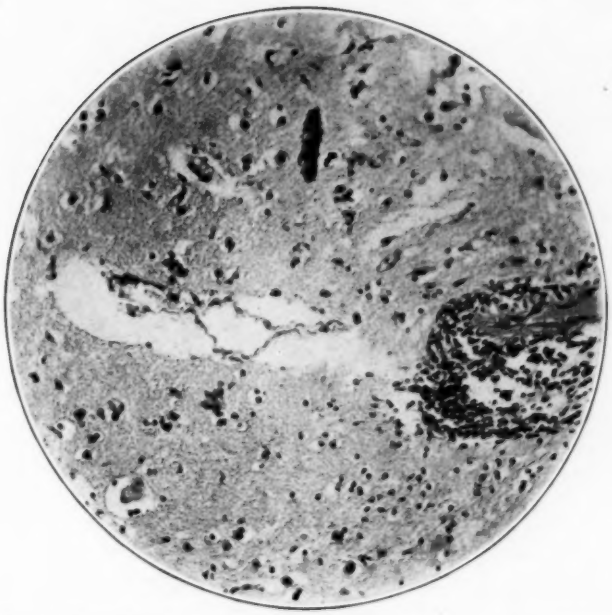

Fig. 8 (case 3).—Caudate nucleus, showing marked infiltration of vessel just below lateral ventricle. There is increase of round cells in tissue.

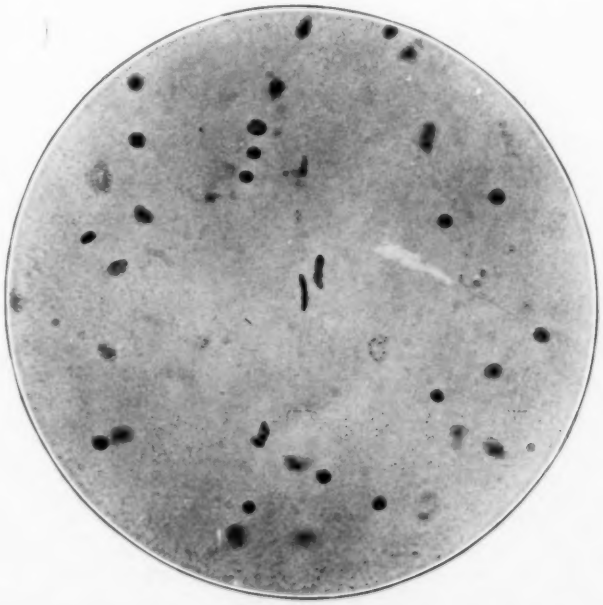

Fig. 9 (case 3).—Caudate nucleus, showing rod cells and plasma cells.

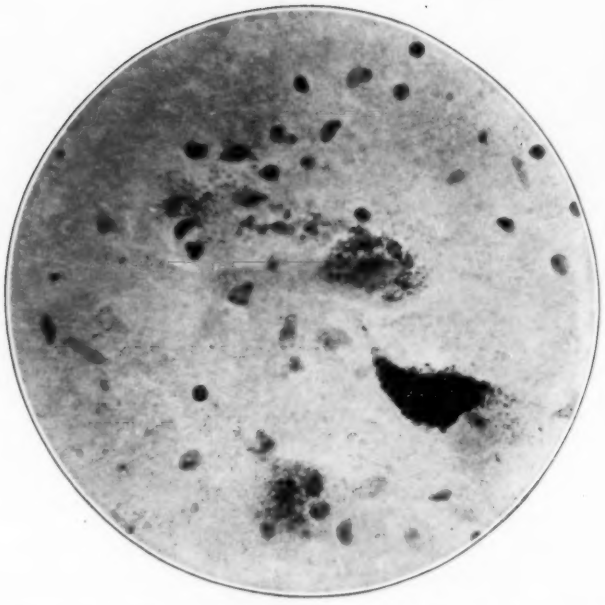

Fig. 10 (case 3).—Substantia nigra, showing marked degeneration of two nigra cells; one cell completely destroyed; pigment scattered. Phagocytosis can be seen, with one cell fairly normal.

Fig. 11 (case 3).—Corpus callosum, showing slight perivascular infiltration.

The large motor polygonal cells showed evidence of acute and chronic degeneration. There appeared to be a dropping out of an occasional cell. There was slight general increase in neuroglia fibers. Pathologic changes appeared more severe in the inner than in the outer segment.

Optic Thalamus: Vascular changes were similar to those found in the caudate nucleus. Rod cells and proliferating glia cells were numerous. Acute and chronic degeneration of the large sensory cells was present; the acute changes semed to predominate; gliosis was present. Satellitosis of three and four glia cells, neuronophagia and occasional phagocytosis were present. Cells bulging with excessive amount of lipoid substance were seen.

Red Nucleus, Nucleus of Luys and Substantia Nigra: These nuclei showed only slight perivascular infiltration with round cells; vessels were seen with

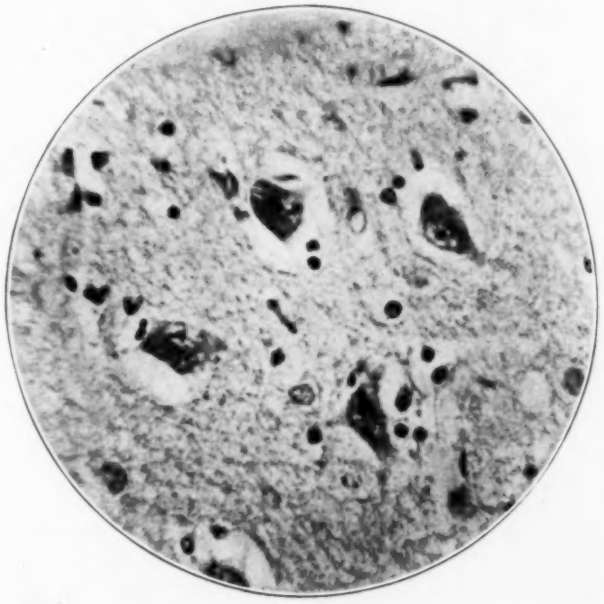

Fig. 12 (case 4).—Optic thalamus showing, degeneration of large sensory cell; collection of round cells about one cell; satellitosis.

only slight changes; new blood vessel formation was apparent. There was some acute and chronic degeneration of the large and small stellate cells of the red nucleus and of the large polygonal cells of the nucleus of Luys; marked granular degeneration of some cells was present and severe disintegration of a cell was occasionally observed. Changes in the substantia nigra were of about the same extent.

Dentate Nucleus: A few small lymphocytes and an occasional plasma cell were seen about some of the blood vessels. Rod cells and proliferating glia cells were present, but not numerous. Occasionally a large dentate cell was seen undergoing the process of acute degeneration; excentric nuclei and granulation were encountered in a few cells. New blood vessel formation was occasionally seen.

Corpus Callosum: Only a few vessels showed slight infiltration with small lymphocytes and plasma cells. Rod cells and proliferating glia cells were present, and there was also slight gliosis.

Case 4.—*Macroscopic Examination.*—Marked convolutional atrophy of the frontal and parietal lobes with thickening of the pia-arachnoid was present, and

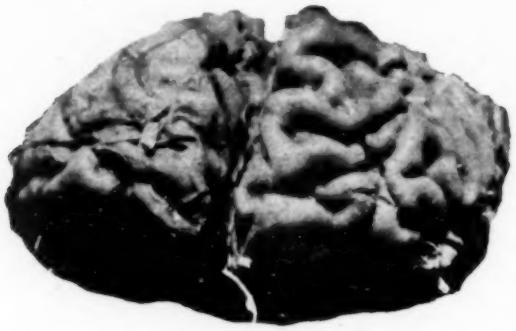

Fig. 13 (case 1).—Frontal lobes, showing marked cortical atrophy; widening of sulci; thickening of pia-arachnoid.

Fig. 14 (case 3).—Brain, showing thickening of pia-arachnoid; atrophy of convolutions of frontal pole; thickening of dura.

was most marked over the same areas. In the widened sulci between the convolutions, there was a thick gray gelatinous substance. The vessels showed moderate arteriosclerosis.

Microscopic Examination.—Cortex: The pia-arachnoid was definitely thickened, because of edema, fibrosis and round cell infiltration. The cortex was

markedly altered; it had lost its orderly arrangement, and the cells were placed in a haphazard manner, many having disappeared; those that remained showed degenerative changes. The vessels, especially the veins, showed perivascular infiltration with lymphocytes and plasma cells. Rod cells were present.

Caudate Nucleus and Putamen: There was marked perivascular infiltration with round cells, mostly small lymphocytes and plasma cells, about all vessels. This infiltration, however, was more severe in the caudate nucleus than in the putamen, and in the margin of the caudate bordering on the ventricle it was more severe than elsewhere. The intima of many of the vessels was broken and irregular; new vessel formation was everywhere present. Proliferating glia cells and rod cells were abundant. There was fairly marked degeneration, more acute than chronic in type, of the large and small stellate motor cells; marked pigmentation appeared in many cells. Ballooning and granulization were seen in many cells. Satellitosis and neuronophagia of four and five cells were seen. General gliosis was present. All cell changes were more severe in the caudate than in the putamen; in the portion of the caudate near the ventricle the cell changes were more severe than elsewhere; the cellular architecture was disturbed, and cells appeared to have dropped out.

Globus Pallidus: Perivascular infiltration and vascular changes were not as marked as in the caudate nucleus. Rod cells and proliferation of glia cells were marked. Chronic, but mostly acute, changes of the large motor ganglion cells were present, many of the cells containing a large amount of pigment; marked granulation was present. Occasionally a cell appeared to have dropped out.

Red Nucleus, Nucleus of Luys and Substantia Nigra: Perivascular infiltration was present, but not nearly so marked as in the other structures described. Acute and chronic cell changes were present; shrunken elongated dark staining cells were frequently seen; other cells showed all stages of acute degeneration. Rod cells, gliosis and proliferating glia cells were present. Neuronophagia of three and four glia cells was seen. Chromatolysis of cells occurred.

Dentate Nucleus: Slight perivascular infiltration was seen about some, but not all, vessels; there was no noticeable thickening of the walls or increase in endothelial cells. The infiltration consisted mostly of small lymphocytes, but an occasional plasma cell was seen. A few rod and proliferating glia cells were present. Many large dentate cells showed evidence of various stages of acute degeneration.

Corpus Callosum: Perivascular exudation of a few round cells was occasionally seen. Rod cells and proliferating glia cells were present.

SUMMARY

The uniformity and consistency of the cellular pathologic change found in the basal ganglia in ordinary cases of general paralysis may be noted. It would also be of interest to theorize concerning these observations in explaining symptoms found in advanced cases; similar symptoms, usually more severe, are found in nonspecific diseases of the basal ganglia.

CONCLUSIONS

1. Definite pathologic changes occur in the basal ganglia in ordinary advanced cases of general paralysis.

2. These changes are similar to and quite as severe as those found in the cortex.

3. The brain tissue bordering the ventricles shows greater pathologic changes than the tissue at a distance from it.

4. The etiologic factors in the origination of the speech disturbance, the expressionless facies and the fine tremors about the eyes and mouth found in cases of general paralysis may be the pathologic changes in the cells of the basal ganglia.

TUBERCULOMA EN PLAQUE*

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Tuberculosis within the cranial cavity is of sufficient frequency to excite interest and to arouse suspicion whenever there is a question as to the causation of a given intracranial disease. Tuberculous meningitis is by far the most common cerebral variety of this disease encountered, and its recognition is not difficult. However, confusion in diagnosis between a focal tuberculous lesion and a brain tumor is exceedingly common; in the instance here presented there was a form of tuberculoma whose rarity makes it of considerable importance.

A brief review of the subject of meningeal tuberculosis would seem to place this lesion, the first described in the English literature, in its proper classification. Tuberculous meningitis occurs as a direct extension of a tuberculous osteomyelitis, especially of the petrous portion of the temporal bone, or by a metastatic infection from the lung or genital tract in adults; from bronchial or mesenteric glands in children, and at all ages from infected bones. In several large series of cases of urogenital tuberculosis in adults, one third of the patients have developed meningeal tuberculosis. Anatomically, this disease assumes one of four forms, the most frequent being the disseminated, associated with much exudation, almost invariably fatal in a few weeks, and involving the leptomeninges of the base and cerebellar surfaces with the greatest frequency. The other three forms are to some extent localized.

The first form consists of a solitary tubercle originating in the pia, but extending into the cortex, varying in size from that of a walnut to that of a goose egg and, according to Oppenheim,[1] exactly simulating in appearance a chronic pyogenic abscess.

The second group is made up of more or less discrete tubercles scattered along the branches of the pial vessels in one region, most often those of the rolandic fissure although cases are described by Saenger[2] and others involving the vessels of the sylvian fissure, and isolated descriptions are found in the literature of symptoms and lesions limited to a portion only of the frontal, paracentral or parietal lobes. Secondary degeneration and softening of the cortex sometimes follow if thromboses occur. Interesting case records are to be found in the

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*From the Medical Service of St. Luke's Hospital.

1. Oppenheim: Lehrbuch der Nervenkrankheiten, Leipzig **2**:890, 1911.
2. Saenger: München. med. Wchnschr. **50**:991, 1903.

literature describing such lesions. A classic example is that described by Busse [3] in a woman, aged 37, who for many years had shown mental disturbances: hallucinations of sight and hearing, an altered personality and a habitus varying from timidity to violent attacks of temper. During the last two years of the illness there were also pains in the head and back, with fever and paranoid ideas. The sputum contained tubercle bacilli. Necropsy disclosed several discrete nodules in the parietal and frontal region adherent to the pia and cortex but separable from the dura. These were flat masses, sharply outlined, but with indented borders. They extended along the central sulcus and onto the medial side of the parietal lobe. They were from 3 to 1.5 cm. in diameter. This case is remarkable for the duration of the symptoms, probably for ten years and certainly for more than two.

Foerster [4] has also described five cases of circumscribed tuberculous meningitis with headache, vomiting and optic atrophy, and a few localizing symptoms—a syndrome that might lead one to believe the lesion a brain tumor. The chronicity of his cases was noteworthy, the duration being from nine months to two and a half years. Necropsy disclosed here also circumscribed areas of scattered tubercles in the paracentral, left central and right frontal regions.

The third type, the so-called méningite en plaque tuberculeuse, was first described by French authors, notably Chantemesse,[5] in 1884. Boiret [6] emphasized the fact that it is a form of cerebral tuberculosis seen only in adults, and described a case showing symptoms of jacksonian epilepsy, headache and vomiting. The patient died in two months, and necropsy disclosed a plaque extending over a large part of the frontal and parietal convolutions. The mass was soft, gelatinous and opalescent, but proved to be tuberculous.

Baumler [7] also described two patients, one observed for seven years, the other for four weeks. These patients showed typical irritative symptoms of the left extremities, and at necropsy the one presented two calcified and the other two caseous tubercles along the course of the rolandic fissure.

Warrington [8] has added an account also of a man, aged 35, ill for only five weeks, with partial paralysis of the face and an upper extremity, and showing at necropsy two plaques on the surface of the left hemisphere.

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3. Busse: Virchows Arch. f. path. Anat. **145**:107, 1896.
4. Foerster: Berl. klin. Wchnschr. **48**:404, 1911.
5. Chantemesse: Étude sur la méningite tuberculeuse de l'adulte, Paris, 1884, cited by Boestrom, in Brauer and Schroeder: Handbuch der Tuberculose, Leipzig **4**:202, 1922.
6. Boiret: Gaz. d. hôp. **72**:390, 1899.
7. Baumler: Deutsche Ztschr. f. Nervenh. **39**:341, 1910.
8. Warrington: Lancet **2**:1754, 1910.

More recently, Czirer [9] has described, with excellent photographs, exactly the type of lesion which we are here presenting, though situated in the region of the sylvian fissure—a mass about the size of the one under discussion, 8.5 by 5.7 cm. in length and width, and 0.5 cm. thick. The clinical history here also is interesting, the patient being a man, aged 25, giving a history of having had fifteen "fits" during the preceding three years. The acute and terminal portion of the illness was characterized by headache, fever and restlessness, which continued for two months.

It will be seen that the differential diagnosis presents several difficulties closely correlated with the anatomic picture. The lesion is essentially localized and meningitic without exudation—even an excess of protein is not necessarily present; the ocular fundi have been described as normal more often than otherwise; the course varies, but has been heretofore noted as lengthy—certainly of seven years' duration and probably more. The gross lesion is so remarkably localized and may be so solid a mass, without the calcified or caseous areas so characteristic of tuberculosis, that tumor is first thought of, especially when the lesion is in contact with the dura. Czirer noted the tumor-like appearance and states that only the circumscribed and noninfiltrating character of the plaque differentiated it grossly from carcinoma or endothelioma. Oppenheim also refers to the almost constant tendency of large meningeal gummas to be greatly flattened and warns against a differential diagnosis being made in some cases without the isolation of the tubercle bacillus. Clinically, fever is almost constantly present in these cases and often constitutes the best guide to diagnosis. Signs of pulmonary tuberculosis or other focal lesions may not be recognizable.

The case presented in this paper was watched from its incipiency with an intermission to its termination, for a period of five years. Its symptomatology of jacksonian attacks was sufficiently confusing in its earlier stages to make a positive diagnosis impossible, while the physical signs of right-sided hyperparesthesia and hyperreflexia were so variable that the inevitable conclusion was made that we were dealing with a hystero-epilepsy.

REPORT OF CASE

History.—In May, 1919, the patient had the first convulsion. This lasted one-half hour; there were numbness and spasm of the right hand, pronation of the hand and loss of consciousness. The patient awoke with a severe headache. There was a second convulsion six months later, similar to the first except that the face was drawn to the right side and there was involuntary urination and considerable prostration. Repeated attacks occurred thereafter. The patient was admitted to St. Luke's Hospital, Feb. 15, 1920, in the service of Dr. Samuel W. Lambert, to whom we are indebted for the privilege of reporting the case.

9. Czirer: Centralbl. f. allg. Pathol. u. path. Anat. **25**:152, 1914.

Course.—The past and family histories were not noteworthy. During this residence, several attacks were noted with the following characteristics: a warning, such as a dull ache in the right arm and hand with headache; numbness and paresthesias of the hand preceding the onset by about two minutes. Then there was a clonic flexion-pronation spasm of the right arm and wrist, with weakness of the grip and arm. With this there was no loss of consciousness; rightsided hyperreflexia was noted. A transitory facial weakness was present. Careful laboratory study at this time revealed nothing abnormal except a slight anemia. The ocular fundi were normal; the serology was normal, and the neurologic examination gave negative results, except for the variable deviations already mentioned.

Although the patient was discharged from the hospital with the diagnosis of jacksonian epilepsy, she was recommended for further observation at the Neurological Institute because we felt that a focalized lesion was possible. She remained under my (I. P.) care there for two months. About ten days after admission, the patient complained of spasticity of the right side of the face, arm and legs; she said that she was going to have another attack; she felt some pain and numbness of the arm to touch and on extension. There was headache on the left side, frontal and occipital. The pupils were dilated; the tongue deviated slightly to the right; there was some weakness of the right side of the face. The right arm was weaker than previously and reflexes were greater in the right arm than in the left. The left upper abdominal reflexes were sluggish. The knee reflexes were: right, 4 +; left, 3 +. The ankle reflexes were: right, 4 +; left, 3 +. There was a tendency to clonus on the right and once an inexhaustible clonus was obtained. There was left plantar flexion. The right toes separated from each other with a tendency to a Babinski sign.

Examination by Dr. Holden at no time revealed papilledema, but on several occasions the staff were convinced of uncertainty of the disk outlines and slight elevation.

During the patient's stay in the Neurological Institute the temperature was normal, except for an occasional rise to 100. The laboratory examinations were repeated, and no variation was found. March 27, 1920, the nurses' notes said that the patient called loudly for the doctor, saying that she was going to have an attack; she kept on talking, saying that she could not let go of the bedclothes with the right hand, and would soon lose consciousness. Clonic movements of the right arm occurred, at first slight, then of increasing severity; the head turned a little toward the left; the mouth was open; breathing was stertorous toward the end of the attack; throughout the attack the eyes winked when touched with cotton. No definite loss of consciousness occurred. The next day she had severe headache and was unhappy and miserable.

The patient was discharged in May, 1920, because there was not considered at that time sufficient evidence to justify an exploratory craniotomy; the hysteroid elements of personality were so marked as to mask somewhat the organic picture. The diagnosis on discharge was, however, a questionable cortical tumor.

The patient next came to St. Luke's Hospital, in March, 1925, and died two days later. The intervening history is of interest. After discharge from the Neurological Institute she improved. Later, however, she went to Bellevue Hospital where a history was obtained of failing eyesight and ringing in both ears for four years. The attacks had occurred at about two week intervals, similar in character to those already described, with no loss of consciousness, but preceded by violent headache and followed by sleep and occasional vomiting.

Six weeks before admission, the patient began to lose control of the right arm and the right side of the face. Examination showed marked choked disk in both eyes, with swollen veins; there was internal strabismus of the left eye; the tongue deviated to the right; the right reflexes were greater than the left. There was no Babinski sign or clonus. The spinal fluid was under increased pressure. The patient was again removed to St. Luke's Hospital, but death

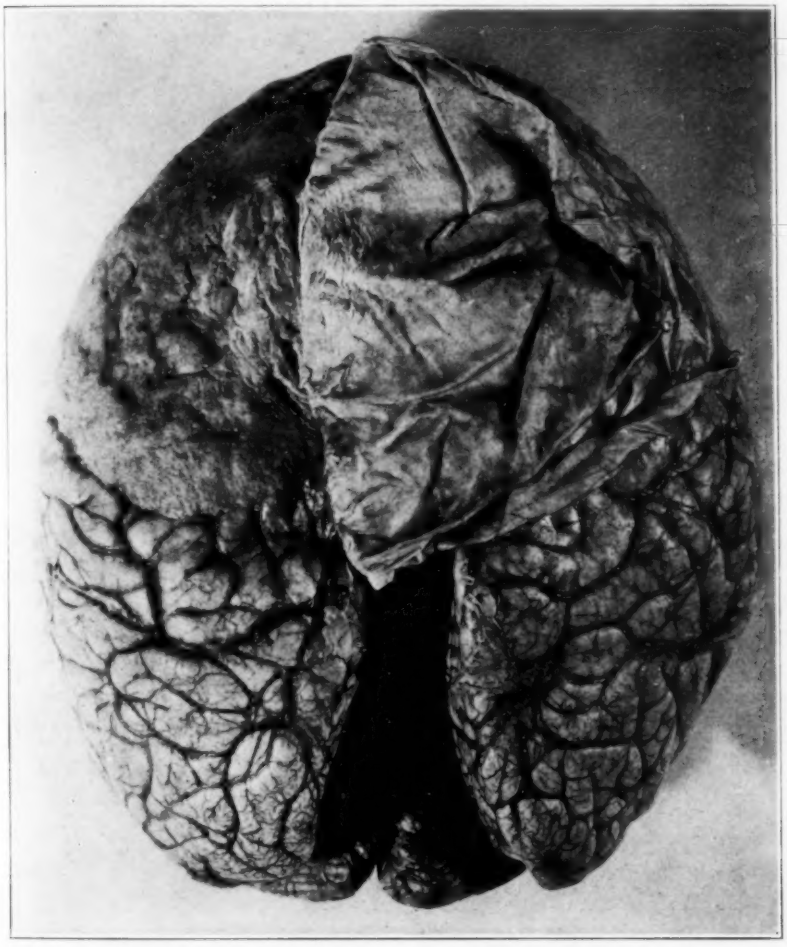

Fig. 1.—Brain with dura reflected from the left frontal cortex to show the cortical plaque.

occurred the day after admission from respiratory paralysis. The antemortem diagnosis was a left parietal tumor.

Necropsy.—The following observations were made: The surface of the left hemisphere presented a large, flat plaque, 9 cm. long and 8 cm. wide, the anterior pole a little narrower than the posterior. This was adherent to the dura near the longitudinal fissure in which the pacchionian granulations were obliterated by the dense scar tissue. The dura could therefore be removed only by cutting

it from the medial half of the plaque (fig. 1). The mass was grayish yellow, scarcely differing in color from the rest of the cortex; it was raised above the surface hardly more than 2 mm., and was very firm, with a finely granular surface and a delicately crenated margin, with no fibrous capsule. At the extreme edges the plaque could be lifted up from the underlying cortex, which it appeared very slightly to overlap, but as a whole it was inseparable from the cortex, which appeared extensively infiltrated by it. It covered the left

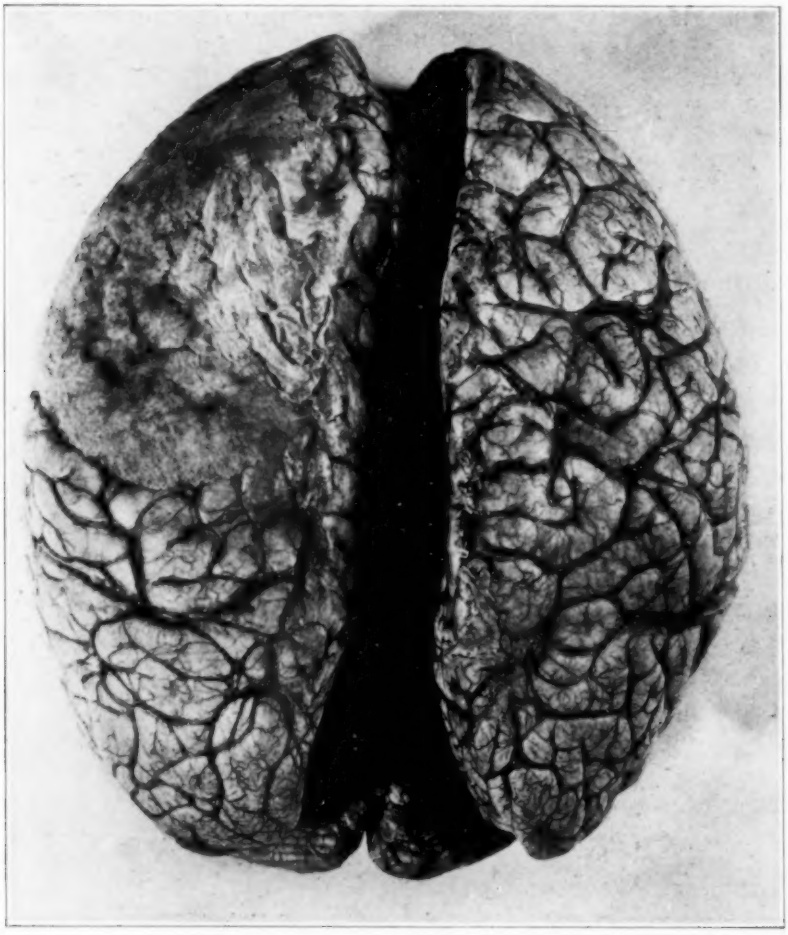

Fig. 2.—The cerebral hemispheres after removal of the dura, showing the large flat plaque in the left frontoparietal region.

superior frontal gyrus, most of the mesial frontal, the anterior and posterior central gyri, and the anterior part of the superior parietal region (fig. 2). It spread to the mesial surface of the hemisphere to the distance of 1 cm. It varied in thickness from 0.3 to 1 cm., the thickest part being near the center of the plaque. The gray cortex was merged with the plaque, the two forming a very hard yellowish mottled but fairly homogeneous mass (fig. 3). No calcified or softened areas were discovered. The mass appeared quite avascular, and did

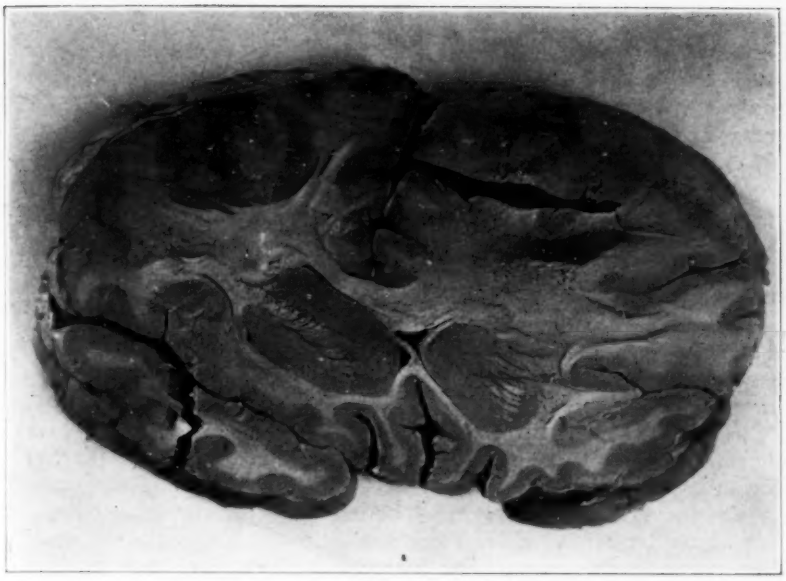

Fig. 3.—Frontal section of the brain through the middle of the plaque showing the gray matter in the frontal cortex replaced by the tuberculous tissue (viewed from in front).

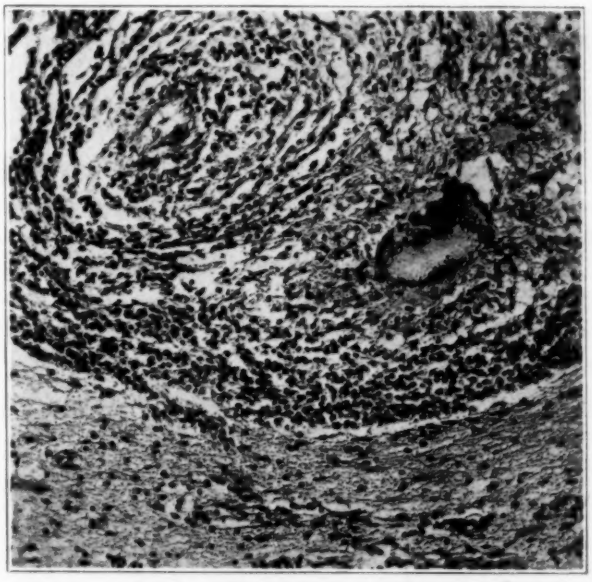

Fig. 4.—Appearance of the frontal cortex and tuberculous meningeal tissue. The latter contains two tubercles with endothelial cells and typical Langhans' giant cell in one. The exudate is chiefly of round cells.

not show evidence of old hemorrhage with pigmentation. Section of the brain showed the ventricles to be of normal size, the meninges elsewhere entirely normal, and the ependyma and choroid plexuses also normal. Frozen sections from the lateral ventricles around the venae terminales and from the choroid plexus of the lateral ventricles as well as from the septum pellucidum showed no thickening or tubercles. The meninges of the base, those of the sylvian fissure and those from the dorsum of the cerebellum showed no evidence of tuberculosis or other inflammation.

Sections through the plaque showed a dense hyaline, fibrous reticulum containing innumerable tubercles. These varied in character and age from fibrous ones near the surface to typical active tuberculous lesions with a caseous matrix, Langhans' giant cells and a peripheral border of endothelial and round cells. The round cells were especially numerous in the deeper layers of the pia, and in some sections a distinct separation of it from the cortex was evident, showing the tuberculous granulation tissue almost entirely limited to the meninges. Within the sulci there were fewer tubercles, and the exudative reaction was moderate. Many round cells were, however, within the cortical substance (fig. 4). In other sections, those from the thickest median portion, there was no line of demarcation, and the cortex was extensively invaded. There was a little thickening of the wall of one of the largest veins, but elsewhere there was almost no change in the vessels, nor was there any tendency for the tubercles to involve them especially.

Stains of the tissue for tubercle bacilli were negative. In spite of this (and the fixation of the brain for this purpose was not the best), it seemed highly probable that the lesion was a tuberculous one. Such well formed tubercles and numerous Langhans' giant cells are not seen in syphilis; nor was there anywhere a suggestion of a syphilitic vascular lesion, either perivascular or subendothelial.

COMMENT

The course of this patient's illness was characterized by the definite jacksonian quality of the convulsions, ushered in first by sensory symptoms and then a definite tonic spasm. Apparently because of the very superficial cortical involvement, hardly penetrating through the gray matter, the disturbance of the reflexes was slight, as shown by the repeated observations by different examiners; the reflexes were variable in their intensity with a generalized hyperreflexia and, if anything, a tendency to exaggeration on the right side. This was noted especially after the attacks. Inexhaustible clonus was only once obtained on the right side, and a Babinski sign was never surely elicited until the last admission when the patient was practically moribund.

The hysterical personality of the patient, which so dominated the picture, and the retention of consciousness and her personal interest in the attacks led us to postpone a more positive diagnosis. Because of this she became lost to our observation, returning too late for operative procedure.

The macroscopic appearance of the tumor was so suggestive of a glioma at necropsy that until sections were examined it was so considered, until the well-known giant cell reaction of tuberculosis was observed.

Clinical and Occasional Notes

IRRESISTIBLE MASTICATION DURING EPIDEMIC ENCEPHALITIS*

PAUL J. ZENTAY, M.D., ST. LOUIS

I have been unable to find in the literature on epidemic encephalitis any report of cases in which there occurred violent and irresistible masticatory movements of sufficient extent to result in severe self-injury. The case here reported seems to be unique and is of exceptional interest because it throws light on the problem of the existence and location of a subcortical center for mastication.

REPORT OF CASE

Clinical History.—G. C., a boy, aged 13, was admitted to the St. Louis Children's Hospital, Dec. 26, 1924, with a history of slight fever and increased drowsiness for the preceding ten days.

Physical Examination.—At the time of admission the temperature was 38 C. (100.4 F.). The patient was deeply lethargic and could be aroused only with difficulty. Questions were answered fairly rationally, but in a slow, dragging fashion. There was a general tendency to muscular rigidity and catatonia, and the face was typically masklike. There was horizontal nystagmus. The neurologic observations otherwise were negative. The spinal fluid was normal.

Course.—During the first two or three weeks in the hospital the patient became progressively more lethargic; it was difficult to get any response except by the most energetic stimulation, and then usually only a wailing, animal-like cry. At times the patient's usually mask-like face became distorted and showed a vivid expression of distress. During the fourth week of the illness the patient slowly became less lethargic, and there were short periods when he was able to answer questions. By the fifth week he could give a fairly good account of his condition. He stated at this time that he had been conscious during the entire illness, and remembered all that had been going on around him.

At no time did there seem to be any disturbance of sensory innervation. According to the patient's statement he had suffered from severe pain when he was subject to involuntary movements that resulted in self-injury.

There was no evidence of involvement of the pyramidal tracts, but voluntary movements of the extremities became more and more difficult without increase in rigidity. This difficulty disappeared during convalescence. There was weakness of the peroneal muscles on both sides, which developed during the second week of the disease and showed no tendency to improve until several months later, when restoration of function was complete. This symptom seemed to be due to a peripheral neuron lesion.

The initial rigidity increased to an extreme degree during the fourth and fifth weeks and then slowly, but totally, disappeared. The catatonia, which was present at the onset, increased during the first three weeks but was not readily

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demonstrable during the period of extreme rigidity. Speech was at first slow and difficult; later, verbigeration developed, and the patient repeated the same words over and over. Still later there was violent shouting, successive words often being uttered with a crescendo intonation. During the third and fourth weeks, there was profuse perspiration limited to the face and neck. Transient polydipsia occurred during the same period.

In general, the psyche of the patient was the usual one seen during the course of epidemic encephalitis. There was partially preserved consciousness, with retarded reactions, extreme slowness of voluntary movements and "difficult breaking through of volition."

Compulsive Attacks.—During the fifth week of the illness, the patient started to suck his lips almost constantly, and the left side of the cheek was at times sucked in between the teeth. When the cheek was brought between the teeth violent chewing motions occurred. To prevent injury, a nurse was in constant attendance, and a padded tongue depressor was held between the teeth. Despite these precautions, the patient chewed his left cheek so as to cause a tear, about 3 cm. long, extending to the left angle of the mouth. A dental wax splint was tightly fixed between the teeth and cheek, and the jaws were closed with a tight pressure bandage. The patient's violent masticatory movements promptly broke the splint and chewing of the cheek continued as before. The tongue was also badly injured. Finally a vulcanite-aluminum splint was made and fixed by wires to the lower jaw. This was effective in preventing injury to the cheeks but seemed to provoke more violent chewing movements. So violent were these movements that a number of teeth were broken off.

The chewing movements came in attacks with temporary remissions. The attacks were usually worse during the night. Each attack lasted from one-half hour to several hours, during which the movements were ceaseless. The attacks continued for two weeks and then gradually ceased.

During the period of the chewing attacks there was little change in the general psychic condition, which was clearing at the time the attacks began. The patient had then improved to the point of answering questions rationally and fairly readily, and there were no signs of clouded consciousness or disorientation. He seemed fully aware of what he was doing and that he was causing serious injury. When asked why he was chewing, he answered that he could not help it. He was aware of the approach of an attack, of which he would inform his attendants, and showed signs of discomfort and pain; but he did not show these signs while chewing the lips or when the teeth were broken.

The patient was discharged from the hospital, April 24, 1925, four months after admission, when he had recovered completely except for the injury to the mouth and jaw, and a slight bilateral footdrop that disappeared later. At this time the intelligence quotient was 106, and the patient gave the impression of brightness with keen interest in the environment.

Fourteen months after discharge, when this report was written, the condition was unchanged.

COMMENT

Undoubtedly, this was a case of epidemic encephalitis, but it presented an unusual combination of symptoms. In addition to the usual hypertonic and hypokinetic symptoms, extrapyramidal rigidity and lack of muscular movement, there was coordinated hyperkinesis in a special group of muscles. This combination would not be so unusual if it were not for the fact that the hyperkinesis was limited to the masticatory muscles and that it took the form of an irresistible compulsion, which resulted in self-injury.

The two American monographs on epidemic encephalitis, that of Tilney and Howe,[1] and the "Investigation by the Association for Research in Nervous and Mental Diseases,"[2] as well as the German monograph by Stern,[3] give no clue to the interpretation of this case. Wexberg[4] has analyzed disturbances of mastication and deglutition in epidemic encephalitis, but even his rich material and theories fail to explain its pathophysiology. Some of his conclusions will be used as a basis for the analysis of this case.

Exner[5] gives a clear analysis of the function of mastication and points out that the masticatory muscles are moved more instinctively than consciously, and are innervated from both hemispheres almost equally so that unilateral lesions rarely cause disturbance in mastication.

Wexberg[4] sums up the physiology and pathology of mastication as follows:

> The question of the location of a center for mastication and deglutition has not yet been answered. The reflex acts of sucking and swallowing in an infant are essentially medullary reflexes. . . . The functions of chewing and voluntary swallowing, on the other hand, are acquired readily and are not primarily of reflex but of automatic nature. . . . The primary automatisms of chewing and swallowing are possible even with bilateral destruction of the cortical centers. . . . It seems necessary to assume the existence of a subcortical center for these automatisms. . . . The function of the hypothetic subcortical centers, being superior to the bulbar nuclei as side connections, will be the eliciting of automatic succession of movements with simultaneous blocking of the bulbar spastic reflexes. Therefore, a release blocked by bilateral cortical lesions will not manifest itself if the automatic centers and their centrifugal tracts are intact. While the blocking function of the cortex, as far as automatic succession of movement is concerned, is taken over by the subcortical centers, the motor function of the cortex cannot be substituted. Cortical paresis, therefore, may lead to disturbance of the automatic succession of movements, but this will be less significant than the disturbance of conscious-voluntary movements for the reason that the subcortical centers by themselves are able to inhibit the reflex spasms. The reverse condition, i. e., serious disturbance of automatic movements with relatively intact voluntary innervation, practically never occurs. This is explained on the supposition that the cortical centers will insure the performance of movements even in case of complete destruction of subcortical centers. . . .
>
> Three possible locations may be considered for the explanation of the disturbances of mastication and deglutition in the different forms of encephalitis: (1) Bulbar, characterized by fibrillary twitchings, atrophy and changes in the electrical excitability of the muscles which appear equally paralyzed for conscious-voluntary, automatic and reflex impulses. (2) Supranuclear, in the pons, pedunculi, corona radiata and bilaterally in the cortex. This is characterized by a typical pseudobulbar form of paresis often combined with spastic-paretic symptoms in the extremities. (3) Basal ganglia in which the pseudobulbar paralysis is combined with disturbances of movements of extremities of an extrapyramidal character.
>
> Whether the disturbance of automatic functions of mastication and deglutition in a like case was caused by cortical paresis or by an involvement of the subcortical centers cannot be definitely decided with the present knowledge. The lesion may be in the corpus striatum or in its centrifugal tracts. All three forms occur in encephalitic processes.

This analysis of the function of the cerebral centers of mastication and deglutition gives a useful basis for the explanation of my observations. The most important parts of the analysis, from my point of view, are the remarks concerning the subcortical center. This "center," while it differs from the cortex, is in control of the succession of automatic innervations of the masticatory muscles. Wexberg arrives at the conclusion that one must assume the existence of such

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1. Tilney, F., and Howe, H. S.: Epidemic Encephalitis, New York, Paul B. Hoeber, 1920, pp. 252.

2. Acute Epidemic Encephalitis: An Investigation by the Association for Research in Nervous and Mental Diseases, New York, Paul B. Hoeber, 1921.

3. Stern, F.: Die epidemische Encephalitis, Berlin, Julius Springer, 1922, pp. 228.

4. Wexberg, E.: Schluck- und Kaustörungen in epidemischer Encephalitis, Ztschr. f. d. ges. Neurol. u. Psychiat. **71**:210, 1921.

5. Exner, S., in Lehrbuch der Physiologie, edited by Trendelenburg, 1924, p. 685.

a subcortical center to explain the symptoms observed. In all his cases there was paresis of the masticatory centers; in my case there was no sign of paresis. I agree with Wexberg, nevertheless, that the existence of such a subcortical center is probable. The symptoms cannot be explained by a center at the bulbar level with nuclear innervations analogous to the anterior horn ganglion cells. If the center were at the cortical level the symptoms could be explained to some extent, but closer analysis of the disturbance will exclude both bulbar and cortical levels as possible sites of the lesion. Only through a subcortical center is it possible to account satisfactorily for the unusual disturbances observed in my case.

The next question that arises is: What is the nature of the disturbances of innervation? This disturbance manifested itself in involuntary movements and in the form of irresistible and uncontrollable attacks. According to the present knowledge, this can be explained either as a release phenomenon or as an excitation of certain centers. "Release" means paresis or disconnection of a higher nerve center leading to loss of control or inhibition of a lower center. This, in my case, would mean paresis of the subcortical and cortical centers of mastication and would lead back to the symptomatology analyzed by Wexberg, i. e., mainly bulbar spasms; but, in my case, none of the usual symptoms of paresis and spasms were observed, so that unless some imaginary and improbable side connections are assumed for the masticatory innervations, release as a possible explanation of the disturbances must be abandoned.

It becomes necessary, then, to assume an excitation at one of three possible levels. Two of these, bulbar and cortical, can be easily excluded as the symptoms produced by excitation at these levels are well known. Bulbar excitation would cause tetanic contraction of the muscles but would not produce succession and automatism of movements. Definite exclusion of cortical involvement is difficult, but if cortical irritation were the cause of the disturbance one would expect that the form of the seizures would be different from those observed. In my case the attacks were limited to the small territory of the masticatory muscles; if cortical in origin, they would have to be classed as jacksonian seizures. It is improbable that motor attacks produced by cortical excitation would last for several hours without apparent loss of consciousness, and also that, with consciousness unimpaired, the attacks would escape voluntary influence. If the excitation were cortical, one would expect it to spread to other areas of the cortex. Furthermore, cortical excitation, as a rule, leads to tonic-clonic movements of epileptiform character. From these considerations, it seems probable that the excitatory process was located in subcortical centers and in the extrapyramidal system. Little is known as yet regarding the occurrence and the characteristic features of excitatory processes in this region, and it is only such observations as those reported here that will throw light on the subject.

The excitation and increased susceptibility to stimuli must have been caused by the virus of epidemic encephalitis, which may have been either a toxic effect or an actual lesion of the subcortical centers. It is impossible to explain the selective predilection for the masticatory center, and the production of entirely different symptoms in the rest of the extrapyramidal system.

Several features of the involuntary movements seen in my case deserve closer analysis: 1. The movements were not continuous but occurred in paroxysms. This probably was due to summation of stimulation in the centers involved, leading finally to a discharge of tension. 2. The attacks of chewing seemed to be independent of voluntary innervation or of stimulation from the periphery and so appeared to be due to endogenous causes in the masticatory centers themselves. 3. Neither ataxia nor incoordination, such as is often seen

in other forms of involuntary movements, was present. In fact, the movements presented all the characteristics of complete succession and automatism of innervation such as occurs in normal chewing; yet, in spite of this, they were lacking in purposefulness. 4. The movements were unquestionably not subject to voluntary control. At the same time they were not below the threshold of consciousness. 5. The independence of the involuntary innervations was sufficiently extreme to act against the integrity of the body. Severe self-injury was the result.

In view of these facts the movements in question could not be classified as voluntary muscular responses. They can hardly be grouped with known forms of involuntary movements of extrapyramidal origin, which appear as pulsions, akathisia, torsion spasms, etc. Such disturbances always involve a large number of muscles and usually those serving the functions of locomotion. It is possible that an important automatism such as chewing takes a somewhat different position among the subcortical centers and in the extrapyramidal system; my observations may indicate that the extrapyramidal innervation and the subcortical center play even a more important part in the automatic functions of mastication than Wexberg's analysis would suggest. The involuntary movements in my case seem to approximate more nearly some of the tics and compulsions observed in psychoses.

My case also suggests several other problems as yet unsolved:

1. Can a subcortical or other nerve center acquire, under any conditions, such far-reaching independence as was observed in this case? Here were observed not only involuntary and uncontrollable attacks but an actual revolt of one center against the welfare and integrity of other parts of the body. In this respect the symptoms are analogous to those observed in some psychoses, with the essential difference, however, that there were no other signs of psychic disturbance.

2. Did pain accompany the tissue destruction? The patient was apparently conscious all the time and gave answers to questions; he could even foretell the approach of attacks. He complained of pain in different parts of the body but not in the jaws or cheeks. This suggests that analgesia, limited to the mouth, existed, but I could find no anesthesia or analgesia with the usual methods of examination in other areas supplied by the fifth nerve. If analgesia was present, it differed from that usually observed.

3. Was the absence of pain sensation due to temporary interruption of conductivity in the sensory paths, possibly in the optic thalamus, or was it produced by diversion of attention by unusually strong stimuli originating in other centers of the brain? If so, why was pain absent in the intervals between attacks? The breaking down of the usual defense mechanism of the nervous system caused by absence of pain must have been one of the essential factors in the disturbance, as pain, if present, probably would have prevented the extensive self injury.

4. Why was the compulsory mastication accompanied by sucking the lips and cheeks between the jaws? This certainly does not belong to the normal mechanism of chewing.

5. Was the patient fully conscious throughout the attacks? By the usual signs, he was fully conscious. It is difficult to explain the fact that he was unable to check the impulse despite the fact that he was conscious, aware of an approaching attack and realized the serious self-injury being inflicted. A few weeks after the attacks, the patient had no recollection of what had happened during the critical days and could give no account of his actions. This may mean that, in spite of the apparently clear consciousness, he was in a clouded state, at least for the compulsory acts of mastication.

SUMMARY

1. In a case of epidemic encephalitis, with typical symptoms during the first four weeks of the illness, during the fourth and fifth weeks, the patient developed attacks of violent compulsory mastication in which he injured the lips and cheeks by biting them.

2. Complete recovery occurred without motor, sensory or psychic residuals.

3. Analysis of the symptoms points to the existence of a subcortical center for mastication.

News and Comment

AMERICAN NEUROLOGICAL ASSOCIATION

The fifty-third annual meeting of the American Neurological Association will be held in Atlantic City, N. J., on Tuesday, Wednesday and Thursday, May 24, 25 and 26, 1927.

BRITISH-AMERICAN NEUROLOGICAL MEETING

A joint meeting of the section of neurology of the Royal Society of Medicine and of the American Neurological Association will be held in London, England, on July 25, 26 and 27, 1927. The program will be published in the next issue.

Obituary

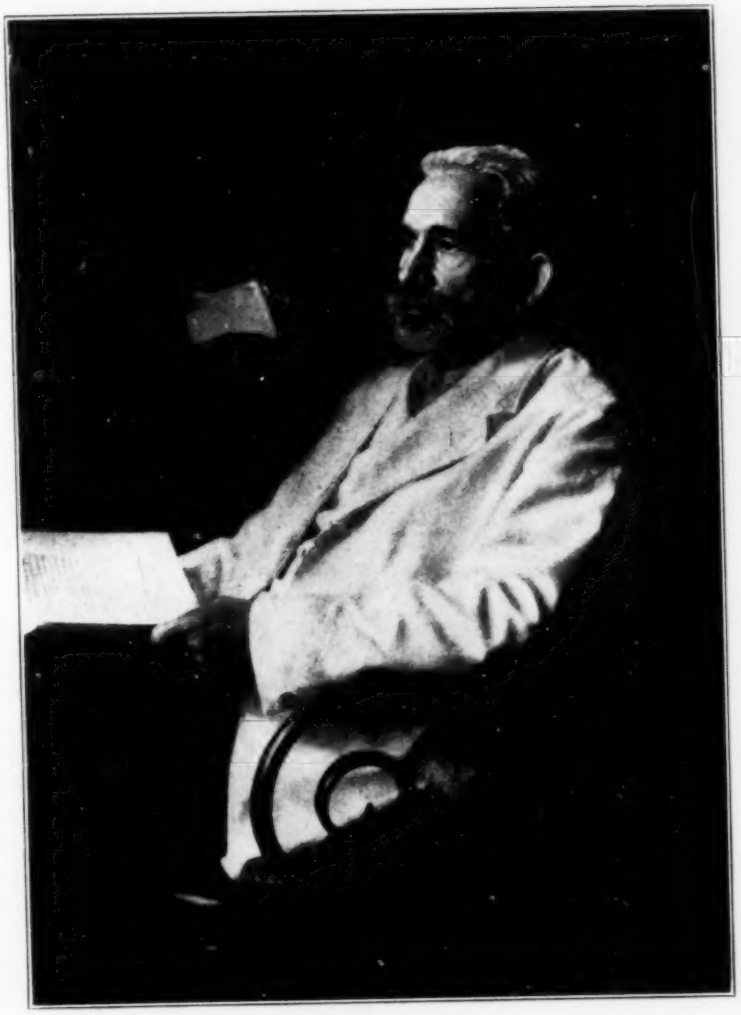
EMIL KRAEPELIN, M.D.
1856-1926

Not quite two years ago Prof. Emil Kraepelin with his associate, Professor Plaut, visited the United States. Although 69 years old, Kraepelin showed an unusual vigor in his quest for data on general paralysis in the white and the colored races of this country. He was full of determination in his plans for new developments in his Forschungsanstalt and no one would have thought of his sharing the fate of Nissl, Brodmann and Alzheimer, that of being carried off at the very beginning of a new era of unrivaled opportunities. An intestinal upset brought on a cardiopathy and caused an unexpected, rapid death, Oct. 7, 1926.

Kraepelin's career, unlike that of most of his continental colleagues, was that of a systematic pursuit of psychiatry, free of any neurologic preoccupations. From his last medical semesters, he turned to Rinecker of Würzburg and Gudden in Munich and when the neurohistologist, Flechsig, from Ludwig's laboratory, was given the chair of psychiatry in Leipzig, Kraepelin became the psychiatrically trained assistant, with a sound clinical background and a keen desire to extend his field in the direction of psychologic laboratory work under the egis of Wundt. He soon broke with Flechsig and devoted himself to studies on the influence of drugs and alcohol on mental functions; in 1883, he dedicated to von Gudden the first edition of his Compendium der Psychiatrie, destined to become, in its eighth edition, the most comprehensive presentation of psychiatry ever written by any one writer.

His marriage caused financial problems which made him accept a position in a nonacademic state institution until good fortune brought him the professorship in psychiatry at Dorpat, and with it a chance to develop his clinic with a psychologic laboratory and a number of enthusiastic students. His textbook grew, and his studies of the work-curve, sleep and drug-effects account for the welcome reward of the successorship of Fürstner in Heidelberg, and a timely escape from the Russification policy in Dorpat. This was the beginning of the "Heidelberg School," which he developed with the collaboration of Ashaffenburg, Nissl, Alzheimer, Gaupp and others, with a brilliant record of literary and scientific productivity. The death of Bumm in Munich brought to him the call to the nearly completed clinic which he presided over from 1904 until he withdrew to the exclusive directorship of his Forschungsanstalt.

An unflinching energy and determination is back of this career. An uncompromising relation toward his contemporaries and, in the suc-



EMIL KRAEPELIN, M.D.

1856-1926

cessive editions, toward his own work gave a personal but firmly authoritative and never hesitating stamp to his work. The swing from a descriptive psychiatry to a prognostic policy created a nosologic supremacy which, to my mind unfortunately, impressed itself on the whole world, while the experimental psychologic work dropped behind and hardly ever attained any fundamental applications in his clinical psychiatry. The final organization of a superinstitute of research with Spielmeyer, Plaut, Rüdin, Jahnel, in the face of the loss of the three giants of neurohistology and neuropathology (Nissl, Brodmann, and Alzheimer) and the devastation of the war, constitutes a height of aspiration and attainment unequalled in the history of psychiatry in any country.

The interests of Kraepelin were those of a strictly theoretical-dogmatic pursuit of a certain type of experimental method—the method of continued work and the work-curve in contrast to the momentary test, reaction-time and argumentation—and, in the clinical field, of the nosologic development of disease entities and their synthetic description. Apart from the early study of the psychoses as related to infectious diseases (1887), he did not create any monographic clinical studies outside of his textbook, and only in his later clinical lectures did he give one an insight into his method of study and utilization of specific cases. The fame of Kraepelin came through the abolition of the concept of secondary dementia and the demand and promise of a prognostic diagnosis along the lines of Morel's and Kahlbaum's formula for a "disease": a specific cause, course and outcome and a specific lesion. General paralysis was his paradigm, and although even that entity yielded extraordinary fluctuations in the statistics of the Heidelberg clinic—with obvious imperfection of the "clinical" diagnosis—the application of the principle to the nondeteriorating recurrent and to the progressive deteriorating psychoses was too enticing not to figure generally as an actual solution of the "psychiatric misère," as Wernicke called it. With rare honesty Kraepelin himself describes how, in the course of the enthusiasm, extraordinary high-water marks of diagnoses of excessive general paralysis and dementia praecox were reached. The 30 per cent of the admissions for general paralysis shrank again to about 10 per cent with Sicard's spinal fluid technic, and the Wassermann reaction; and the 51 per cent of admissions diagnosed in one year as dementia praecox were reduced to a more probable 20 per cent or less in the course of time, especially through the recognition of the importance of the affective traits and perhaps more attention to the all important constitutional component. At the time when, during the war, the American Medicopsychological Association legislated the dogmatic assumption of the "either-or" of manic-depressive insanity or dementia praecox, Kraepelin already had approached the less dogmatic concept

of reaction types. It seems highly probable that the attention to developments from specific initial factors will become more and more significant; but the perspective of certain trends culminating in various degrees and types of deterioration always will be an item in psychiatric reasoning, and its connection with Kraepelin's name will be indelible.

There is a long step between the beginnings and the end-stages of the developments that today are called dementia praecox. Attempts to fill them with freudian dogmatism have been fought persistently by Kraepelin. It is, nevertheless, a task made mandatory even with Kraepelin's insistence on the dominant importance of the end-result that the intermediate steps from the beginnings to the ends and the nature of the "process" be worked out, whether one sticks to Kraepelin's large group entities or to a freer, more genetic-dynamic conception of mental disorders.

The Kraepelin school was strikingly remote from the interest in the study of the individual patient and was bent on generic principles. This has had a great economizing value, but it may easily be that the best study will have to be one of a number of simpler units of functions than what is emphasized in the kraepelinian mass-problems. Thus the heredity studies, in the hand of Kraepelin's assistant, Kahn, already have led to the assumption of two components in dementia praecox, a constitutional and a process factor, whatever this may mean.

Kraepelin was one of the most ardent protagonists against the alcoholization of Germany and since the war had been an equally ardent nationalist. His property near Pallanza had been confiscated by Italy. In the face of the merciless, nationalistically colored criticism of Kraepelin's work by Lugaro, the Italian government restored to him the home in which he spent six months of the year while he gave the other six months to the Research Institute, the further development of which had been made possible by a grant from the Rockefeller Foundation. Two large volumes of psychiatric papers constituted the two "Festschriften" devoted to the celebration of his seventieth birthday.

The record of Kraepelin always will remain a great stimulus in psychiatry. He may have unduly overshadowed in his time various other movements; but during a period when medicine was too exclusively interested in infections and disorders of the functions of simpler organs, he was one of the few capable of securing a hearing for psychiatry. His work did much toward balancing extremists and will continue to make for critical reserve within the ranks of the more judicious workers in the field.

ADOLPH MEYER.

Abstracts from Current Literature

THE CHEMOTHERAPY AND SERUM THERAPY OF PNEUMOCOCCUS AND STREPTOCOCCUS MENINGITIS: 1. A RÉSUMÉ OF THE PRESENT STATUS OF THE TREATMENT OF SEPTIC MENINGITIS, WITH THE RECOMMENDATION OF A METHOD. JOHN A. KOLMER, in collaboration with ANNA M. RULE and BERNARD MADDEN, Arch. Otolaryngol. **3**:481 (June) 1926.

Acute suppurative pneumococcus, streptococcus and staphylococcus meningitis, of otitic or nasal origin, or following fractures of the base of the skull, shows a mortality of 100 per cent. An occasional case with recovery has been reported. Some of the reported recoveries probably have been due to the meningococcus. Kolmer has seen two cases of alleged pneumococcic meningitis in which recovery took place. His examination showed the presence of the meningococcus in each.

Pneumococcus meningitis developing in the course of pneumonia without discoverable foci in the upper respiratory tract has a slightly better prognosis. Cases of meningismus are not included in this paper, which deals only with active suppurative cases. Treatment in these cases by spinal drainage and intraspinal injection of antistreptococcus serum and their intraspinal and intravenous use has produced no results. The following investigations were made to determine the value of various forms of treatment for these highly fatal cases: Highly virulent cultures of a type 1 pneumococcus and hemolytic streptococcus were used. Rabbits were found unsuitable on account of the frequency with which fatal bacteremias developed. White rats and guinea-pigs were too small, so dogs were used in order to determine the effects of lavage and drainage. Dogs of from 5 to 10 Kg. were used. Cultures were injected into the cisterna. Generally, symptoms of meningitis were apparent within twenty-four hours after infection with pneumococci, but were sometimes delayed up to four days in the case of streptococci. Treatment was generally delayed until symptoms appeared. Untreated controls usually died within five days after the onset of symptoms. All treatments were given under ether anesthesia, and the animal was kept under the influence of morphine and provided with special care. Cases in which the dose did not produce the fatal meningitis in the controls were excluded.

The Chemotherapy of Septic Meningitis.—In the present series of experiments, no recoveries were observed in the dogs with experimental pneumococcus meningitis that were treated with intravenous, intracisternal or intraventricular injections of ethylhydrocuprein. The dose of ethylhydrocuprein hydrochloride by repeated intracisternal or intraventricular injection with safety cannot be much more than from 12 to 15 cc. of 1:1,000 solution for an adult weighing 130 pounds (59 Kg.), although by intraspinal injection a somewhat larger amount may be given after removal of a larger amount of spinal fluid.

Ethylhydrocuprein Hydrochloride in the Treatment of Pneumococcus Meningitis.—It was found that injections not later than twenty-four hours after infection showed a slightly curative effect; in virulent cases in which the injection was delayed twenty-four hours, there was no effect. Mixtures of ethylhydrocuprein and homologous antipneumococcus serum or mixtures of the drug serum and sodium oleate did not yield better results. The daily or twice daily withdrawal of spinal fluid, followed by the intraspinal injection of from

12 to 20 cc. of from 1:500 to 1:1,000 solutions in saline solution may prove of aid in the treatment of pneumococcus meningitis in man. The author has treated eleven patients with pneumococcus meningitis during the last six years. All the cases were of this origin. In three, life was prolonged, and in one hope of recovery was entertained. Recoveries have been credited to the administration of ethylhydrocuprein, chiefly in cases of pneumococcus meningitis developing during pneumonia in which spontaneous recovery is possible and the prognosis much better than in cases developing secondary to primary otitic or nasal infections in which the mortality is practically 100 per cent. It is in these cases in which Kolmer has used ethylhydrocuprein by intraspinal injection combined in some cases with intravenous injection, followed by oral administration to raise the bactericidal activity of the blood.

In the present investigation, delayed intravenous injection of ethylhydrocuprein hydrochloride had no effect, and daily puncture of the cisterna magna with drainage of spinal fluid followed by injection of the drug was without effect. Intraventricular injection, sometimes combined with intravenous or intramuscular injections of the drug, gave negative results. Somewhat more encouraging results were obtained at times when the administration of ethylhydrocuprein was combined with pneumococcus antibody solution. The author believes that these results were due more to the antibody solution than to the drug. Negative results were observed after the use of type 1 antipneumococcus serum. He concludes that a daily intraspinal intracisternal and intraventricular administration of ethylhydrocuprein, in a dose approximating 0.0002 Gm., or about 15 cc. of a 1:1,000 solution per 130 pounds, is without effect in the presence of acute pneumococcus meningitis. It is also without effect experimentally when the first dose is delayed after early symptoms have appeared.

Summary.—"1. The daily intraspinal, intracisternal or intraventricular injection of ethylhydrocuprein hydrochloride in doses of 0.0002 Gm. per kilogram had no appreciable curative effects in the treatment of acute experimental pneumococcus meningitis of dogs. This dose corresponds to from 12 to 15 cc. of a 1:1,000 solution per 60 Kg., or about 130 pounds.

"2. No better results were secured by combining these routes of administration with the intravenous or intramuscular injections of the compounds.

"3. Combined treatment with antipneumococcus serum and ethylhydrocuprein did not improve the results, although some improvement was noted in combined treatment with pneumococcus antibody solution.

"4. Especially good results were secured by lavage from the ventricles to the cisterna magna, followed by intraventricular and intracisternal injections of the drug, but lavage alone has yielded similar results."

Mercurochrome.—Intravenous injection of mercurochrome-220 soluble had no appreciable effect on the course or mortality of experimental pneumococcus and streptococcus meningitis in dogs. The same was true when the drug was administered by intracisternal or intraventricular injections. Mercurochrome may aid in the removing of the bacteremia, which sometimes develops in meningitis. It has proved of some value by intravenous injection in the treatment of severe mastoid infections. Gentian violet, neutral acriflavine and a flavine dye were used with some degree of local bactericidal influence but with no duration of effect on the life or mortality.

"Chemotherapy alone, therefore, has little or nothing of real value to offer today in the treatment of severe septic meningitis, if treatment consists of

the usual method of draining the purulent cerebrospinal fluid and injecting ethylhydrocuprein, mercurochrome, gentian violet, neutral acriflavine or a flavine dye subthecally or intravenously. It does not appear possible to introduce sufficient by subthecal injection to influence materially the infections, and it is doubtful if intravenous injections are capable of actually affecting the destruction of organisms in the infected meninges and cerebrospinal fluid. But if means are provided for draining away the purulent cerebrospinal fluid, and especially for lavage of the ventricles and subarachnoid space in general, we believe that the administration of these agents, and particularly of ethylhydrocuprein and the dyes, may be useful as adjuvant measures."

Mercurochrome-220 Soluble in the Treatment of Pneumococcus and Streptococcus Meningitis.—The toxicity of mercurochrome for the meninges and its relatively low bactericidal activity for pneumococci and streptococci and in purulent spinal fluids soon indicated that it may not be of value in these conditions. The author's attention was called to one case in which death followed a gross error in dosage—20 cc. of a 1 per cent solution having been injected intraventricularly, with a prompt lethal result. Mercurochrome in purulent spinal fluid shows little beneficial results because of the dilution that it is necessary to use.

Summary.—"1. The maximum tolerated dose of mercurochrome for normal dogs by subthecal injection is approximately 0.001 to 0.002 Gm. per kilogram of body weight, corresponding to 1 to 2 cc. of a 1:1,000 solution per kilogram. The tolerance of dogs with streptococcus or pneumococcus meningitis is less, and by repeated injections the maximum tolerated dose was found to be from 0.0001 to 0.0003 Gm. per kilogram. In the treatment of meningitis by intraspinal, intracisternal or intraventricular injection of mercurochrome, these doses would correspond to 6 to 18 cc. of a 1:1,000 solution (not 1:100) for an adult human being of approximately 130 pounds weight.

"2. The bactericidal activity of mercurochrome for pneumococci and streptococci in purulent cerebrospinal fluids was found to be, in final dilutions, varying from 1:400 to 1:1,000 in an exposure of five minutes at 37 C. In view of the small amounts that may be safely administered by subthecal injection, it would not appear possible, therefore, to secure sufficient bactericidal effects in the subarachnoid space to influence the disease materially.

"3. Mercurochrome in dose of 0.005 to 0.008 Gm. per kilogram by intravenous injection was without appreciable effect in the treatment of streptococcus meningitis of rats, staphylococcus meningitis of rabbits, and streptococcus and pneumococcus meningitis of dogs. Subdural and intracisternal injections of the compound in doses varying from 0.0001 to 0.0003 Gm. per kilogram were likewise ineffective in the treatment of these various kinds of experimental septic meningitis.

"3. Mercurochrome by intravenous injection of maximum amounts may aid in removing bacteremia (septicemias), but is believed to be without curative properties in the treatment of the meningeal lesions of septic meningitis."

Gentian Violet, Acriflavine and a Flavine Dye in the Treatment of Pneumococcus and Streptococcus Meningitis.—Gentian violet and neutral acriflavine have not yielded good results when employed alone in the treatment of experimental pneumococcus and streptococcus meningitis in dogs, although better results were observed when the dyes were used after lavage and in conjunction with serum therapy. A flavine dye has been employed in Germany intravenously in

doses of from 15 to 30 cc. of a 1:1,000 solution for adults of average weight, in cases of streptococcic infection. The chemical purity of these drugs affected their toxicity.

The tolerance of rabbits and dogs with acute streptococcus and pneumococcus meningitis was less than that of normal dogs. The dose corresponded to from 6 to 18 cc. of 1:1,000 solutions per 130 pounds.

The results with gentian violet were of a negative character in the treatment of experimental staphylococcus meningitis of rabbits.

Gentian violet was found to be of no value in experimental streptococcus or pneumococcus meningitis when administered by subdural, intravenous or intracisternal injection. Intravenous injections were of no value, but after the intracisternal injections a local bactericidal infection was noted. Animals treated with antistreptococcus serum and gentian violet gave negative results. Gentian violet was without appreciable effect in the treatment of meningitis of dogs by intravenous and intracisternal injections.

Acriflavine has proved ineffective in the treatment of experimental streptococcus meningitis of dogs, when given by intravenous and intracisternal administration.

A flavine dye gave negative results. The only encouraging results were observed among dogs treated daily by lavage followed by intracisternal and intraventricular injection of the drug. The author believes that the benefit was due to the lavage.

Summary: "1. By injection into the cisterna magna of normal rabbits, the maximum single tolerated dose of gentian violet (Coleman and Bell) was found to be from 0.001 to 0.003 Gm. per kilogram. The dose of neutral acriflavine (National) was 0.002 to 0.003 Gm. and a flavine dye (rivanol, Hoechst) about 0.0005 to 0.001 Gm. per kilogram, by the same route of injection. The tolerance of dogs with acute streptococcus and pneumococcus meningitis, however, was somewhat less and by repeated intraspinal, intracisternal and intraventricular injection, the usual tolerated dose of the three compounds was from 0.0001 to 0.0003 Gm. per kilogram. These amounts correspond to 0.006 to 0.018 Gm., or 6 to 18 cc. of 1:1,000 solutions per 60 kilograms of weight.

"2. The bactericidal activity of gentian violet, acriflavine and a flavine dye for streptococci and pneumococci in purulent spinal fluids varied from a final dilution of about 1:1,800 to 1:4,000 in an exposure of five minutes at 37 C.

"3. Gentian violet, neutral acriflavine and a flavine dye, by intravenous administration in dose of 0.005 Gm. per kilogram, showed no beneficial effects on the course of experimental staphylococcus, streptococcus and pneumococcus meningitis.

"4. Gentian violet, neutral acriflavine and a flavine dye, by intracisternal and intraventricular injection in doses varying from 0.0001 to 0.0004 Gm. per kilogram, had no appreciable effect on the course and mortality of acute experimental streptococcus and pneumococcus meningitis of dogs."

The Serum Therapy of Septic Meningitis.—Four patients with type 1 pneumococcus meningitis treated with injections of serum with boric acid and sodium oleate were not improved.

The intramuscular, intravenous, intracisternal and intraventricular injection of polyvalent antistreptococcus serum, including a concentrated serum, failed to influence appreciably or constantly the course and mortality of meningitis

in dogs produced by intracisternal injections of the authors' culture of highly virulent hemolytic streptococcus.

The same has been true in the treatment of experimental meningitis in dogs produced by intracisternal injections of virulent type 1 pneumococci and treated with intramuscular, intravenous, intracisternal and intraventricular injections of stock polyvalent or type 1 antipneumococcus serum.

In the experiments on dogs, "intramuscular, intravenous, intracisternal and intraventricular injections of antibody solution alone have not saved the lives of any of our animals, but the duration of the lives of several has been prolonged for a few days, and when administered along with lavage of the ventricles to the cisterna magna, complete recoveries have occurred. This would appear to be a particularly desirable product for the treatment of meningitis when it is desirable to inject subthecally as powerful a product as possible in the least bulk of fluid."

Lavage and Drainage in the Treatment of Septic Meningitis.—Certainly it is one of the outstanding conclusions that drainage alone or with lavage constitutes the most important phase of treatment. Drainage as described by Dandy is worthy of serious consideration, even though subdural irrigation, as originally advised by Eagleton, recently has been abandoned by him.

"In our experiments, the only effective method of treatment found so far in the treatment of severe streptococcus and pneumococcus leptomeningitis of dogs has been trephining and washing one or both ventricles through to the cisterna magna with warm saline or Ringer's solution, escape of the fluid being provided for by the insertion of a needle into the cisterna magna. One treatment alone of this kind has saved animals when the untreated controls have all died in the usual time of four to seven days. Sometimes pus continued to drain from the trephine opening for a few days, and these animals usually fared better than those without this drainage; we believe that lavage alone is full of great promise in the treatment of septic meningitis of human beings, provided treatment is instituted sufficiently early in the disease. We have been able to save dogs by this means after the onset of symptoms but before the exudate became too purulent and plastic."

Recommendations in the Treatment of Septic Meningitis.—The examination of the spinal fluid is necessary in making a diagnosis. The author "feels confident that the slow and careful removal of a few cubic centimeters of cerebrospinal fluid from a child or an adult suspected of having meningitis will do no harm either in the way of favoring the localization of organisms in the meninges from the blood, or by favoring the extension of a localized meningitis, and that this kind of spinal puncture should be resorted to early for diagnostic purposes."

"If the fluid is found clear, one would naturally hesitate to inject anything subthecally," although it is well, in the author's opinion, to inject antibody solution intravenously or subcutaneously in cases of suspected pneumococcus meningitis, or mercurochrome-220 solution or gentian violet solution intravenously in suspected streptococcic infections.

If a purulent fluid is recovered, indicating the presence of widespread leptomeningitis, he believes the case should be regarded at once as one requiring surgical intervention, calling for the drainage of the local focus of infection if present or discoverable, the performance of trephining and puncture of a ventricle, together with puncture of the cisterna magna and lavage from the ventricles to the cistern. He has usually washed out the dogs with from 20 to 40 cc. of warm saline or Ringer's solution, and has never observed any ill

effects. Lavage from the cistern to the lumbar region of the cord may be done at the same time, although he is convinced that septic meningitis kills from the cistern upward rather than by spinal meningitis. Washing out the ventricle on the infected side of the head is recommended, although in severe cases both ventricles may require irrigation. In pneumococcus infections, lavage may be followed by the introduction of pneumococcus antibody solution in doses of about 0.5 cc. for each 2 pounds of weight. Whether or not several treatments of this kind will be required he is unable to state; this, doubtless, will depend on the clinical status of each case; but with infected dogs one treatment has usually sufficed. At all events, he hopes that surgeons will not hesitate to institute treatment along these lines under ether anesthesia, particularly in view of the fairly well established clinical and experimental fact that ordinary treatment by spinal puncture and intraspinal medication is almost sure to fail.

Immune Serums, Antibody Solution and Lavage in the Treatment of Pneumococcus and Streptococcus Meningitis.—The intraspinal injection combined with intravenous or intramuscular injections of antistreptococcus or antipneumococcus serums has proved to be of no benefit. The author has treated seven patients who had streptococcus meningitis with polyvalent antistreptococcus serum administered by intraspinal and intravenous injection, with no recoveries. In pneumococcic meningitis of otitic or nasal origin the mortality has been 93 per cent. Occasionally recovery occurs in a case of serous meningitis and sometimes in a case in which pneumococci are found in cultures with clear fluid without leukocytic exudations. He has treated patients who had otitic pneumococcus meningitis with intraspinal and intravenous injections of polyvalent antipneumococcus serum, with recoveries. In four of these the intraspinal injections were of mixtures of antiserum sodium oleate and boric acid. The prognosis in cases of pneumococcus meningitis occurring in lobar pneumonia is better than in cases of otitic or nasal origin. Kolmer used fresh sterile normal chicken serum by intraspinal injections in doses of from 8 to 10 cc. in two cases without beneficial effects. He is sure that autoserum therapy would not prove effective.

Pneumococcus antibody solution as employed by Wieder by intraspinal injection has given hopeful results. Wieder's patient rapidly improved, only to succumb to an unsuspected abscess of one of the frontal lobes of the cerebrum. It is the most promising biologic product available at present for treatment of pneumococcus meningitis.

Lavage of the ventricles and subarachnoid space is the most valuable means of treatment, especially when drainage from the cisterna magna is provided. Dandy has reported the recovery in three of four cases of septic meningitis ascribed to provision for continuous drainage from the cisterna magna. Two of these were streptococcus infections and one *Staphylococcus aureus* meningitis. The fatal case was caused by hemolytic streptococcus. In experimental meningitis, he found lavage of the ventricles to the cisterna magna the most beneficial of all treatments. He has continued this study along the lines of establishing continuous drainage from the cisterna magna and spinal meninges combined in some instances with lavage by way of the ventricles and subdurally by means of trephining. The results will be reported later.

Antistreptococcus Serum in the Treatment of Streptococcus Meningitis of Dogs.—Stock concentrated polyvalent antistreptococcus serum was used. Treatment was begun twenty-four hours after infection before symptoms had developed or about four days later when signs of meningitis with cloudy spinal fluid were present.

"Under anesthesia, serum was injected intraventricularly and intracisternally in dose of 0.2 to 0.3 cc. per kilogram of body weight, but in no instance were beneficial effects observed." Combination of this treatment with intravenous and intramuscular injections of the serum was without results. Combination of the serum intravenously or intramuscularly with mercurochrome intravenously or gentian violet by intraventricular injection was without beneficial results.

"But when one of the ventricles was first washed through to the cisterna magna by the injection of about 6 cc. of warm saline solution per kilogram of weight, followed by the intraventricular injection of serum, cures were effected. In conducting these experiments under ether anesthesia, a trephine was first made with the introduction of a needle into one of the lateral ventricles; then a needle was inserted into the cisterna magna, followed by drainage of purulent fluid and the slow intraventricular injection of warm saline solution. Each dog of approximately 5 to 10 Kg. weight was given between 25 and 40 cc. of saline in this manner, and the treatment was concluded by injecting 0.2 to 0.3 cc. of serum per kilogram into the ventricle and sometimes into the cisterna magna."

Kolmer believes that the larger part of success attending these experiments is to be credited to lavage. "Lavage alone with warm saline solution from the ventricles to the cisterna magna has resulted in saving a number of dogs infected with our strain of hemolytic streptococcus and type 1 pneumococcus, when all of the untreated controls have perished in the usual time of four to seven days after infection by intracisternal inoculation. In some instances, lavage of one ventricle has been sufficient, but recoveries have followed lavage of either one or both although the latter has been a slightly severer treatment by reason of trephining both sides of the head."

"Probably lavage from the cisterna magna to the lumbar region of the spinal subarachnoid space is helpful, but we have not found this necessary in the treatment of these experimental infections of dogs. We have reason to believe that the institution of continuous drainage from the cisterna magna or lumbar subarachnoid space will still further improve the results, and furnish a method of value in the treatment of septic meningitis of human beings."

Antipneumococcus Serum and Antibody Solution in the Treatment of Experimental Pneumococcus Meningitis of Dogs.—The course of experimental pneumococcus meningitis of rabbits was not influenced appreciably by either type 1 pneumococcus serum alone or in combination with sodium oleate and boric acid. Similar negative results have been observed in the present investigation employing dogs infected with intracisternal injections of type 1 pneumococcus and treated with homologous serum and antiserum administered intravenously, intraventricularly and intracisternally. In one experiment intracisternal injections of ox bile alone and in combination with the serum were of no benefit. Antibody solution, however, appeared to yield a better result although Kolmer did not succeed in saving a single animal with it alone. But when it was given after lavage, with saline solution, the majority of animals survived and lived indefinitely. He attributes these good effects entirely to the lavage.

Summary: "1. Polyvalent antistreptococcus serum administered by intramuscular, intravenous, intracisternal and intraventricular injection has failed to influence appreciably the course and mortality of experimental hemolytic streptococcus meningitis of dogs.

"2. Type 1 antipneumococcus serum administered by intramuscular, intravenous, intracisternal and intraventricular injection has failed to influence appreciably the course and mortality of experimental type 1 pneumococcus meningitis of dogs.

"3. The intravenous, intraventricular and intracisternal injection of antipneumococcus antibody solution has exerted a slight degree of curative activity in the treatment of pneumococcus meningitis.

"4. Lavage of one or both of the lateral ventricles to the cisterna magna has proved effective in the treatment of severe experimental streptococcus and pneumococcus meningitis of dogs. This may be combined with the administration of antibody solution by subthecal injection in the treatment of pneumococcus meningitis, or by the injection of gentian violet in the treatment of streptococcus meningitis."

HUNTER, Philadelphia.

THE NEURALGIA CONCEPT AND THE NATURE OF THE NEURALGIAS. Folke Lindstedt, Ztschr. f. d. ges. Neurol. u. Psychiat. **102**:100 (March) 1926.

Lindstedt advances a new conception of the nature of the neuralgias. In a previous article on the nature of sciatica he came to the following conclusions: 1. The sciatic symptoms are combined with myalgic symptoms in practically all cases in the sural and thigh regions as well as in the gluteal and lumbar regions. 2. In the great majority of sciatic cases peripheral causes may be demonstrated to be of great moment in the causation of the sciatic symptoms. Such causes included not only tumor formations, meningitic and spondylitic processes, coxarthritides, salpingitis, varices, etc., but also less well recognized causes such as flatfoot, genu recurvatum, skeletal anomalies in the sacrolumbar region, ankylotic processes of the vertebrae or of the lower extremities—as in spondylitis deformans or arthritides—shortening or lengthening of either limb, as after fracture, painful or crippling conditions in the joints or soft tissues, as in polyarthritic or gonorrheal arthritis, varices, gangrenous appendixes, hernias, varicoceles and other causes. 3. In isolated myalgic symptoms in the ischial region as, for example, attacks of lumbago, myalgia of the gluteal muscles or thighs occurring without sciatica, it is possible to find causes that are similar to those in sciatica. 4. In brachial, intercostal and occipital neuralgias, peripheral changes and causes, which are responsible for the causation of the neuralgia, may be demonstrated.

Lindstedt begins by asking what is meant by neuralgia. This can be attacked better by asking what concept of neuralgia seems most satisfactorily to explain all known facts. According to the author, none of the present theories of neuralgia is suitable, because none can be carried out logically in practice; he therefore attempts to formulate a new conception. The word neuralgia signifies "nerve pain" and, since every pain is in a sense a nerve pain, should be used for every possible pain. Every pain, moreover, has a localized causative factor; since these pains follow definite nerve trunks and branches, it has been thought that they are due, not to peripherally acting factors, but to disease of the nerves themselves, hence the name neuralgia. "One can therefore say that the original theory of neuralgia included such pains on a definite clinical basis." This is a purely negative conception of neuralgia. However, since neuralgia has also a positive aspect, a positive conception has arisen, according to which a pain is neuralgic if it is elicited by definite nerve changes. The attempt to make neuralgia a disease entity

sui generis gave rise to the etiologic theory that it is a specific disease at the basis of which lie hypothetic, not demonstrable, nerve changes. With this theory an attempt was made to establish a definite symptom-complex characterized by: (1) pains of considerable intensity; (2) their relatively wide distribution and course along nerve trunks and their branches; (3) their lack of continuity but their definite periodicity and occurrence in paroxysms. Some investigators, as Head, Hallion et al., lay most stress on the localization of the pains along nerve trunks; while others, Pitres and Villard and Alexander et al, emphasize the paroxysmal nature. According to Lindstedt, however, the disease entity that should correspond to the etiologic theory of neuralgia symptomatically cannot be differentiated sharply. In other words, there is no such sharply defined clinical picture, for if the idea is accepted that neuralgias are nerve pains dependent on disease processes localized somewhere in the nervous system, it can hardly be said that theories in this form have reference to a definite disease entity. If this theory is accepted, it is true of only a limited number of cases. It has been shown that a number of pains that were looked on as neuralgias, in the etiologic sense, are in fact the result of definite disease conditions. This is true not only of sciaticas, but also of many trigeminal neuralgias occurring as a result of peripheral conditions of various kinds, for example, toothache, sinusitis, otitis, etc. It is true also of certain types of neuralgia of the arm with arthritis of the shoulder joint, as well as of intercostal and brachial neuralgia. These have been separated under the name of secondary or symptomatic as opposed to primary or idiopathic neuralgia. If the occurrence of secondary neuralgia is accepted, then the number of the primary or idiopathic types of neuralgia becomes few indeed. "The better the investigator, the fewer the idiopathic neuralgias." Lindstedt found only nine cases of primary neuralgia among 100 cases of sciatica, and thought these too were probably secondary, though he could not prove it. He believes thoroughly in this concept. Edinger wrote at the end of his life, "The older I get, the more rarely I meet an idiopathic neuralgia."

From the pathogenic standpoint, neuralgia is defined as a pain caused by stimulation of whole bundles of nerve fibers in places in which such fibers are numerous, for example, in the peripheral nerves or dorsal roots (Erb, Sahli, Wertheimer-Solomonson). According to this theory, the neuralgia is a projection phenomenon, the cause of which lies primarily in the nervous system. This theory rests on unstable foundations, for it does not answer such questions as why the neuralgic pains are felt only in the deep parts, why they are found only along the course of nerves, and why they do not call forth disturbances in sensation such as position and temperature. According to the pressure theory, the pain is due to pressure on nerve trunks; but in many instances this cannot be demonstrated, as for example in coxarthritides, anomalies of the extremities, arthritis, etc. These theories play an important rôle in the concept of the neuralgia and are widely accepted. Lindstedt does not subscribe to them. According to Pfeiffer, neuralgia is purely symptomatic; i. e., a pain is neuralgic if it is characterized by certain features such as irradiation, intensity and paroxysms. None of the theories described, etiologic, pathogenic or symptomatic, is satisfactory, because none is logically applicable in practice.

In promulgating his theory, Lindstedt shows that the use of the term neuralgia in designating pains, with or without definite causative factors, follows a definite principle that is based on the inexplicableness of the pains, and which therefore does not coincide with modern principles. For example,

there are many pains with a localizable cause which are not spoken of as neuralgic, although they show a definite neuralgic symptom picture. Such pains occur in visceral organs — angina pectoris, nephrolithiasis, cholelithiasis — and in other bodily parts through inflammatory processes, and one assumes that the demonstrable cause explains the pains completely. If these observations are accepted, then it must be conceded that the neuralgic nature of a pain is a relative characteristic that has no sharp boundary between a neuralgic and a nonneuralgic pain.

Lindstedt outlines his conception of neuralgia as follows: "A pain is more or less neuralgic as far as it appears to stand in one or more definite symptomatic connections, such as intensity, radiation, duration and occurrence, to a localized causative factor or, if this factor is not demonstrable, to the negative characteristics, being related to this factor in a noteworthy and . . . unexplainable disparity." The result of the examination can therefore be positive or negative, and in both cases it is evident that there is an unexplainable disparity between the symptomatology and the localizable etiologic factor. One can therefore state the theory in another way; "A pain is more or less neuralgic in the sense that there is no characteristic pain-producing change or cause that is proportionate to it at the time." The theory itself is negative and has no positive aspect of the nature of neuralgia. It includes such pains as appear to be due to no anatomic-pathologic change or cause. The types of pains included under this conception of Lindstedt include: (1) All pains that are included in present day literature as primary or idiopathic neuralgia. (2) All pains with a demonstrable cause, known as secondary or symptomatic neuralgia. Such pains include the secondary sciatic, brachial, intercostal and trigeminal neuralgias. (3) Most of the so-called muscular rheumatic and myalgic symptoms. These have not hitherto been considered as neuralgias. (4) Many rheumatic pains difficult to classify, such as occur in psychic or other conditions, in temperature increases, weather changes, etc., in all parts of the body but especially in the head, joints and muscles. (5) Painful symptoms emanating from the viscera, as for example in angina pectoris, cholelithiasis, etc. Angina pectoris has been considered a type of neuralgia by some authors (Romberg, 1853, neuralgia cardiaca), but difficulty has arisen with our modern conceptions of neuralgia in the differentiation between neuralgic (angina pectoris spuria) and nonneuralgic pains (angina pectoris vera). (6) Certain pains in a definite part of the body which arise from causes in more or less distant parts — reflex pains.

With regard to the pathogenesis of neuralgia, Lindstedt states that the causes of the neuralgic nature of a pain are in general not to be sought in the causative factor. In fact, the neuralgic nature of a pain is best explained in most cases by factors that are independent of the localized etiologic factor. The etiologic significance of the causative factors lies in the fact that they contain or carry pure peripheral sensory stimuli. The neuralgic pain then becomes a pathologic mode of reaction of the nervous system against sensory stimuli in general. The neuralgic pain would then be an expression of a general nervous disease. Support for this view is found in the fact that general causes are often found in neuralgic pains as, for example, neurotic states, infections and intoxications, diabetes and other metabolic disorders, anemias, cachexias, etc. Neuralgic properties, such as the intensity, radiation and paroxysmal nature of the pains, are in fact nothing more than expressions of a generally increased sensitiveness in the central nervous system against sensory stimuli. Lindstedt would do away with the conception of primary or idiopathic neuralgia, and

would in particular call attention to the fact that a neuralgia is more than a localized expression due to a localizable cause but is really the expression of the entire nervous system to noxious sensory stimuli; that is, it is the expression of a functional change of a general nature in the central nervous system.

ALPERS, Philadelphia.

TIME RELATIONS OF THE VARIOUS PHENOMENA TO THAT OF CONJUGATE DEVIATION IN THE EPILEPTIC ATTACK. A. M. SCHNELL, Arch. f. Psychiat. u. Neurol. **77**:239 (May) 1926.

This is a report of a study of the time relations of the various phenomena observed in the epileptic attack, based principally on a detailed analysis of consecutive attacks in a case of status epilepticus and a comparison between this and other epileptic cases. The author believes that it is best to regard the attack as consisting of eight phases (the eighth being the transition into the interval between attacks). The position of the eyes, with special reference to deviation to one or the other side, is taken as the principal phenomenon, and the time relations between its various phases and those of other phenomena, the condition of the musculature, the secretions, reflexes, etc., are established. Special attention is paid to the significance of deviation in determining the possibility of a jacksonian element in the attack and the possibility of localization of a lesion.

The observations point to the existence of a definite relation of certain symptoms and phenomena to certain time phases of the attack. In the first phase there is conjugate deviation of the eyes and head toward the side that is most affected, and in jacksonian attacks toward the one on which the convulsion begins. With this there are some clonic manifestations with the beginning of the tonic component of the convulsion, gradual decrease of respiratory rate, reddening of the face, swelling of the vessels, and complete areflexia. In the second and third phases, the eyes return to the center and downward; the musculature reaches the tonic stage; respirations are markedly retarded or stopped; the face is blue; the vessels reach maximum engorgement, and there is areflexia. In the fourth stage, there may be conjugate deviation similar to that of the first with nystagmoid jerks toward the same side; with the development of the clonic component, there are increased expiratory movements and the face becomes an earthy color; other phenomena are the same as in the third phase. In the fifth phase, there is the beginning of conjugate deviation toward the side opposite that of the first phase, with solution of the muscular phenomena, deep expiration and pallor (grayish); the vessels begin to return to normal; secretion of saliva and perspiration appears; areflexia is present. In the sixth phase there is complete conjugate deviation to the side opposite that of the first phase; the musculature is weak; breathing is deep and regular and the face is pale; other components are as in the fifth phase. In the seventh phase, there is movement of the eyes upward and to the center; otherwise conditions are similar to those of the sixth phase.

The condition of the pupils is as follows: in the fourth and fifth stages, the pupils are myotic and react to light. The first, second and seventh periods show medium sized pupils and no reaction to light. The third and sixth stages show markedly dilated pupils with no reaction to light. The eighth stage is the one of exhaustion, in which the Babinski phenomenon is most frequently found on the side corresponding to the second conjugate deviation (therefore on the side opposite the one on which the convulsion began or was most marked). This phase leads to that of the interval in which the eyes

are in a position similar to that in the seventh phase: the musculature is weak, though it may show twitchings about the face and eyelids; the respiration is loud; the face is pale and the veins are engorged; the reflexes (in this particular case) were: conjunctival and corneal, absent; Mayer, absent; deep, present; abdominal and plantar, absent.

The author concludes that certain definite time relations can be established in all cases between the various phenomena of an epileptic attack, and that the conjugate deviation of the eyes bears a definite relation to the localization of the point in the brain where the attack first sets in.

MALAMUD, Foxborough, Mass.

PERIODIC OCULAR SPASM IN POSTENCEPHALITIC PARKINSONISM. A. E. KULKOW, Ztschr. f. d. ges. Neurol. u. Psychiat. **102**:636 (June) 1926.

Kulkow reports the occurrence of periodic tonic spasm of the eye muscles causing deviation of the eyeball to its most extreme lateral position. The literature on the postencephalitic eye phenomena is concerned chiefly with permanent eye disturbances—disorders of convergence, spontaneous nystagmus and pupillary disturbances—"while ocular symptoms which are more transitory and occur in paroxysmal form have been only slightly studied." The case described by Kulkow occurred in a man, aged 26, with a typical history of encephalitis. A parkinsonian state came on about six months after the acute attack, and the eye phenomena appeared for the first time about two years after the onset of the illness. The patient complained of inability to blink and of pain in the eyeballs. The eyes moved to the left during attacks. Closing the eyes was still possible, but was performed with the greatest difficulty. During the attack, the eyes could not be moved from a position of extreme fixation to the left; they could not be moved upward, downward or to the right. The arms were held strongly adducted and flexed, and the patient was unable to answer questions during an attack. Marked anisocoria was present during the attacks, the left pupil being larger than the right; reaction to light and in convergence and accommodation was lacking. Usually the attacks lasted two or three hours, but shorter ones lasting about ten minutes also occurred. Though voluntary movement of the eyeballs was impossible during the attacks, in passive movements of the head the eyeballs could move in all directions. At first the attacks occurred only occasionally, but later they took place every three or four days. They usually occurred in the evening. No effect was produced by intravenous injections of methenamine. Atropine caused temporary relief of the ocular spasms, but after a time they recurred with the old regularity. An autoserum injected intraspinally had no effect on the eye symptoms.

Ewald reported six cases of postencephalitic parkinsonism with tonic eye spasms. Bing and Schwartz reported three cases which they described as "vertical oculogyric crises." In these cases the eyes were turned upward. Pappenheim reported three cases of tonic spasm of the eyes upward. A résumé of all these cases shows that the eyes in such attacks can be moved upward, to the right or left; the attacks last from two to three hours; they occur about every four days; sleep puts an end to the attacks, and there is no disturbance of consciousness during the episodes. Kulkow lays great stress on the inability to move the eyes voluntarily in an attack with ability to move them passively with change in the position of the head. Ewald, Schwartz and Bing

compare the attacks to epileptic seizures on account of their periodicity. Kulkow is able to offer no suggestion as to the localization of the pathologic lesion causing the attacks. Most authors ascribe a striate localization on the basis of the tonic character of the attacks, since the corpus striatum is looked on as a tonus regulating mechanism, and with its destruction tonic spasms are observed. The most important argument for a striate localization is the occurrence in cases of paralysis agitans. Bing and Schwartz are in favor of a supranuclear localization on the basis of a similarity of the attacks to an extrapyramidal epilepsy. Sterling has reported ten cases of extrapyramidal epilepsy with tonic spasm in the extremities.

ALPERS, Philadelphia.

THE PROBLEM OF THE SCHIZOID. A. BOSTROEM, Arch. f. Psychiat. u. Neurol. **77**:32 (April) 1926.

The term schizoid, used first by Kretschmer and later by Bleuler, does not seem to imply the same concept invariably. In psychiatric literature, Bleuler's description seems to be the one most generally accepted. Unlike Kretschmer, Bleuler believes that this type of reaction exists to a greater or lesser extent in all human beings, and places it side by side with the "syntonic" reaction (Kretschmer's cyclothymic), which is also found universally. The validity of Bleuler's view is supported by the fact that, in general literature too, the two concepts occur as traits of human nature. Bostroem draws a parallel between Bleuler's concepts of the schizoid and syntonic and Scheffler's description of the Gothic and Greek types, respectively. Starting from different points of view and using different types of material, they reach the same conclusions as to the nature and prevalence of the two types. The schizoid, like the Gothic, is the dissatisfied, progressive, creative type. In contradistinction to the syntonic or Greek type of person, he lacks rhythm and harmony, works in fits and starts, is always dissatisfied with existing conditions and is always looking for something new: the revolutionary type. Scheffler, in observing the different types and periods of art as well as the different periods of history of mankind in general, like Bleuler, sees merely a quantitative and not a qualitative difference in the different types of normal and abnormal persons; now the schizoid and then the syntonic is more marked, but both exist side by side, in all types and epochs.

If one is to accept this concept of the schizoid type and yet preserve the "process" idea in dementia praecox, as Bleuler does, it will be impossible to regard dementia praecox as just an exaggeration of a previous schizoid type. This difficulty will also be found in the manic-depressive group. In fact, the psychologic relations between a schizoid personality and schizophrenia are only superficial; they seem to depend on the fact that the traits of the schizoid personality may be exaggerated by the disease, though they do not represent the symptoms that characterize it. The peculiar mode of behavior of the schizophrenic patient—the loose and dilapidated thinking, the disregard of logic, the projection, in fact, all characteristic symptoms of the disease—has nothing to do with the concept of the schizoid personality itself. The fact that schizoid traits are found in a greater proportion than syntonic traits in the history of schizophrenic patients seems to be dependent on the lesser resistance of the schizoid type to disease in general.

A review of the literature dealing with the study of the prepsychotic personalities of patients with different types of psychoses, as well as of the types

of physique found in them, shows once more that there seems to be no definite basis for assuming any particular type of constitution, either mental or bodily, that corresponds to a prepsychotic stage of schizophrenia. The author therefore concludes that: (1) if the concept of schizoid personality as representing a general character trait is to be accepted, the difference between it and dementia praecox will have to be regarded as qualitative as well as quantitative; (2) no special relations exist between this schizoid type and schizophrenia; (3) in view of these facts, and to avoid misunderstanding, the reaction type described by Bleuler as schizoid and by Scheffler as Gothic should be called dystonic, to contrast it with the syntonic type of reaction.

MALAMUD, Foxborough, Mass.

THE MICROSCOPIC CHANGES IN THE NERVES IN PELLAGRA. N. WINKELMAN, Ztschr. f. d. ges. Neurol. u. Psychiat. **102**:38 (March) 1926.

It is significant that a disease with a nearly typical clinical picture and course should show such varied characteristics in the central nervous system. Poussie, among the earliest workers, found arteriosclerosis and fatty degeneration of the cerebral vessels and nerve cells and concluded that the disease was toxic in nature. Marchi (1888) found poor staining qualities in the posterior horns, and in the fibers of the anterior horns in two cases. Belmondo, on the basis of a study of twenty cases, divided the changes found in pellagra into two classes, acute and chronic. The former is characterized by a meningomyelitis, and the latter by degeneration and sclerosis of the posterior and lateral columns. In addition, he found pigment deposits in the nerve cells, chiefly in the lumbar region, and thickened blood vessels. Tuczek classified the changes in the brain into those occurring as the result of cachexia, and those due to the pellagra itself, such as fatty degeneration and calcification of the intima of the smaller vessels together with pigment deposits in the adventitia. In the cord he found pigmentary changes in the nerve cells and numerous corpora amylacea. Gaucher and Sergent found sclerosis of the lateral columns more pronounced in the lumbar region of the cord, with swelling and fatty degeneration in the ganglion cells of the brain. Lowkoss described leptomeningitis and softenings in the cord as being constant, while other observations were degeneration of the pyramidal and dorsal columns. Babes and Sioli found marked changes in the Betz cells with increase of pigment in the cells of the entire nervous system, changes in the posterior roots and columns, and in Clarke's column as well as in Lissauer's zone. Marinesco found chromatolysis and nuclear changes in the ganglion cells of the cortex as well as in the medulla and cord. Breggia described thickening of the vessels, hyperplasia of the connective substance and decrease in the nerve cells in the ganglia of the sympathetic nervous system. Kazonsky, after a study of ten cases, concluded that the fundamental process is independent of the vascular changes, and that pellagra is a pathologic entity characterized by degenerative changes in the nervous system. Anderson and Spiller found severe changes in the Betz and anterior horn cells with diffuse Marchi degeneration in the spinal cord. The cells of Clarke's column were also degenerated. The and other authors have found many changes in the central nervous system in pellagra.

Winkelman reports three cases of pellagra with necropsy. Two of the cases were typical and showed, on histologic examination, a fatty degeneration of practically all elements of the brain and cord, especially ganglion cells, glia cells, vessel walls, and the ependymal cells of the central canal. Moreover, a

diffusely distributed hyaline degeneration of the small vessels occurred. Besides the fatty changes, the ganglion cells in the cortex and cord showed retrograde changes. A third case is reported which showed, in addition to the changes described above, degeneration in the column of Goll and in a posterior root. The histologic changes form a more or less uniform process, which includes changes in the vessel walls of capillaries and precapillaries, diffuse fatty degeneration of the nervous structure and, finally, changes limited chiefly to the cells of the anterior horns and to the giant cells of the cortex. The question arises as to whether these processes are correlated and what the primary change is. The vascular changes are widespread and may be looked on as a degenerative change dependent on the pellagra. The question as to how far the parenchymatous changes are dependent on vascular processes cannot be answered with finality. The diffuse process seen in the changes in the central nervous system in pellagra speak for the assumption of a systemic disease.

ALPERS, Philadelphia.

PNEUMOCEPHALUS (INTRACRANIAL PNEUMATOCELE OR AEROCELE). WALTER E. DANDY, Arch. Surg. **12**:949 (May) 1926.

Dandy reviews the literature on this interesting condition and describes three additional cases from his own experience. He points out that there are two possible sources of air within the skull: First, and by far the most common, an artificial opening between the interior of the cranial vault and the external air; second, an aerocele formed by gas-producing organisms. A fracture line extending through the paranasal or mastoid sinuses is the most frequent artificial opening through which the air may enter, although operative defects, chronic erosions and destruction of the floor of the skull by tumor or a dilated third ventricle in hydrocephalus may also be factors. A compound fracture of the skull is not enough to produce a pneumocephalus. In order to displace the cerebrospinal fluid, the air must be forced inward under pressure, as may occur in coughing, sneezing, straining or swallowing. Within the skull the air may be found in the subdural or subarachnoid spaces, within the cerebral substance or contained in the ventricle. An air cyst within the cerebral substance is the condition most frequently encountered.

The most usual route of entrance of the air is by way of the accessory sinus subsequent to a fractured skull. Fractures through the paranasal sinuses seem to be followed by a pneumocephalus more commonly than fractures through the mastoid, for the reason that during coughing, sneezing or swallowing more direct pressure is put on the nasal opening and air is thus forced into the cranial cavity. The variety of pneumocephalus is dependent on the sinus involved, the exact character of the fracture and the length of time air has remained in the cranial chamber.

The symptoms of pneumocephalus are principally those of increased intracranial pressure. They may come on immediately or at a long interval after the fracture has occurred. The presence of the aerocele is usually entirely unsuspected. An acute or chronic hemorrhage or abscess is the condition most frequently diagnosed. But if following cranial trauma a cerebrospinal leak occurs, particularly if this escape of fluid is precipitated by or subsequent to coughing or sneezing, and the signs of intracranial pressure develop, the possibility of a pneumocephalus must always be considered. The absolute diagnosis is made by the roentgen ray.

Surgical treatment is indicated to close the aperture through which the air entered the cranium. Especially is this indicated if a cerebrospinal leak exists. The presence of the air in itself is not a source of danger. It is the possibility of infection reaching the subarachnoid space through the compound skull fracture that must be taken into account. To prevent this from occurring, the position of the fracture line and the tear in the dura must be carefully located and an attempt made to close the dural defect by suture or implantation of a graft.

GRANT, Philadelphia.

THE PHYSIQUE OF NORMAL PERSONS. H. W. GRUHLE, Arch. f. Psychiat. u. Neurol. **77**:1 (April) 1926.

In conjunction with Gans of the dermatologic department, the author investigated the physique of 118 persons with skin diseases who, from a psychiatric point of view, could be considered normal. The investigation was carried on along the lines of Kretschmer's types of physique, for the purpose of comparing the results in normal persons with those obtained by different authors on psychiatric material. The material used was mostly from northern Bavaria and southern Baden, and the author suggests that, to confirm his observations, material representative of other regions should be investigated similarly. He concerns himself mostly with the form of the body on the one hand, and with that of the face and head on the other; he then attempts to find correlations between the two. A further point of investigation was furnished by the color of the eyes and hair.

The different types were represented proportionally in percentages as follows: leptosome (asthenic), 20.3; athletic, 18.6; pyknic, 12.7; dysplastic, 11; uncharacteristic, 15.3 and mixed forms, 22. A comparative study of these observations and those of the most important investigations of psychiatric material lead to the following conclusions as regards the proportions in schizophrenic and normal persons: (1) the proportion of pyknic persons is the same; (2) the proportion of athletic persons is the same; (3) there is only a slight difference between the percentage of athletic-asthenic types; (4) the proportion of the asthenic type is the same. The same equality holds true in the dysplastic type so that, constitutionally (if one means by constitution the physique), schizophrenic and normal persons are alike; therefore, schizophrenic persons cannot be looked on as a special constitutional group.

With manic-depressive or cyclothymic persons, the results are somewhat different. There is no characteristic predominance of asthenic persons in either schizophrenia or cyclothymia; whereas the number of pyknic types in normal and schizophrenic persons is the same, manic-depressive persons are characterized by a majority of the pyknic type of physique. On the other hand, the number of athletic types in the cyclothymic group is small.

In general, study of the bodily physique of these normal persons differentiates three definite types; leptosome, athletic and pyknotic. The relation of these types to special forms of face and head, however, is not as definite. The middle-aged pyknic individual is probably the only one that could be said to have a characteristic face form. In the other types, the relations are loose and unreliable.

The author concludes that, whereas the relations between a special pyknic type of physique and the circular psychoses established by Kretschmer probably hold true, there is no reason to assume that schizophrenic persons are characterized by any special type of physique.

MALAMUD, Foxborough, Mass.

THREE TUMORS ARISING FROM NEUROBLASTS. WILLIAM BOYD, Arch. Surg. **12**:1031 (May) 1926.

Boyd describes three cases of tumor in young children arising apparently from the neuroblasts or primitive nerve cells. These tumors may arise in three ways: (1) from original neuroblasts — a neuroblastoma; (2) from the ganglion cells — a ganglioneuroma, and (3) from the chromaffin cells of the paraganglions — a paraganglioma or chromaffinoma. The neuroblastoma proper is by far the most common of these. It is composed of small round cells with dark hyperchromatic nuclei, closely resembling cells of a small round-cell sarcoma. From the latter it may be distinguished by the presence of fibrils that do not give the characteristic staining reaction of collagen, fibroglia or neuroglia. They are arranged in longitudinal bundles and occasionally in rounded balls or masses around which the neuroblasts are grouped so as to give the appearance commonly known as a roset. The absence of rosets, however, does not negative a diagnosis of neuroblastoma. The fibrils must be regarded as naked axis cylinder processes. While they may be stained by Bielschowsky's method, van Gieson's stain gives the most satisfactory results.

While theoretically a neuroblastoma may arise wherever neuroblasts — originally derived from the neuro-epithelial canal — are found, in actual practice they most often occur in the medulla of the suprarenal. The most remarkable feature of these tumors is bone metastases, especially those in the skull. There is a striking clinical picture in young children presented by the association of a tumor of the suprarenal with ecchymosis of the eyelids, unilateral exophthalmos and the frequent appearance of tumefaction in the temporal region. The clinical picture of the child with an apparently bruised face, a protruding eye and lumps over the surface of the cranium is arresting. It is not only in the cranium that bony metastases may occur, but also in ribs, vertebrae, sternum and tibias, while, in addition, there may be involvement of the regional glands, the liver and the lungs.

The three cases described by Boyd were all of this type. One arose from the suprarenal medulla, one from the abdominal sympathetic and the third from the retina. In all these cases the most striking metastases were in the cranium, producing exophthalmos and the bruised effect in the skin over the skull. All the patients died, and the details of the necropsies were carefully given. Pathologically, although originating from different organs and differing in microscopic structure, these tumors nevertheless possess features in common. All may be regarded as developmental tumors arising in children from the neuroblast at different stages of its development. In the first two tumors described, the spread was apparently by the lymphatics; in the third by the blood stream.

GRANT, Philadelphia.

STUDIES ON THE ETIOLOGY OF EPIDEMIC ENCEPHALITIS. ALICE C. EVANS and WALTER FREEMAN, Pub. Health Rep. **41**:1095 (June 4) 1926. (Reprint No. 1085).

Considerable confusion exists in the mind of investigators as to the microbic etiology of epidemic encephalitis. As early as 1917, von Wiesner described a pleomorphic streptococcus which was pathogenic for animals. Other workers have found the same or similar organisms. A large number of negative results has been reported. Other observers have found a virus of the filter-passing group but have not cultivated it. Evans and Freeman were unsuccessful in two chronic cases, but in a third patient dying during a

recurrence of encephalitis an organism was isolated from the nasopharynx during life and from the heart blood and midbrain after death, that was found to be identical with that isolated by von Wiesner and by Rosenow. "The remarkable pleomorphism of the organism suggests life cycles as complex as those of some of the higher fungi. It may be stated briefly that in one of the phases of its life history this organism is a spore-forming rod. The rod form produces not only spores, but also exceedingly minute filtrable, coccoid bodies which develop on the outer walls of these rods. Under certain conditions, these minute coccoid bodies enlarge and multiply as cocci. The detailed study of the rod form and other phases in the life history of the organism will be given in forthcoming publications. This report will be limited to observations on the streptococcus form of the organism—the form in which its virulence is highest and most stable."

The organism developed fairly quickly in fluid mediums, which are described in detail, and later grew on common mediums much in the form of *Streptococcus viridans*. It tended to localize in the brain of rabbits after intravenous injection. It could be transmitted in series, often with augmented virulence. Intracerebral inoculation of 0.25 cc. of a 1:10,000 dilution of a twenty-four broth culture was fatal to rabbits. Monkeys were also susceptible to the disease on intracerebral inoculation. The symptoms observed in the animals were pareses, convulsions, forced movements, peculiar rotations of the head, somnolence and twitching movements of various muscle groups. Necropsy revealed diffuse meningo-encephalitis. Perivascular infiltration was occasionally intense near the aqueduct of Sylvius. The substania nigra did not seem to be seriously affected.

FERRARO, Washington, D. C.

THE PHYSIQUE OF MENTALLY NORMAL AND ABNORMAL CRIMINALS. F. VON ROHDEN, Arch. f. Psychiat. u. Neurol. **77**:151 (April) 1926.

One hundred and fifty mentally normal and ninety-one mentally diseased criminals were examined from the point of view of physique and character along the lines of the Kretschmer types. In both series, different types of criminal acts were equally mixed; but in the mentally abnormal group they were uniformly of a more serious type than in the others. The ages of normal criminals ranged about the third and those of abnormal criminals about the fourth decade. Two thirds of the abnormal criminals developed psychotic manifestations after the crime was committed. As to the type of psychoses, schizophrenia had the largest (60 per cent), and manic-depressive the smallest (4 per cent), proportional representation.

Comparative study of the type of physique of normal and abnormal criminals, on the one hand, and of noncriminal schizophrenic patients, on the other, brings out the following facts: (1) the normal and mentally abnormal criminals show no difference in physique except that the dysplastic type is definitely more frequent among abnormal than among normal criminals; (2) there is no difference between the types of physique of noncriminal schizophrenic persons and criminals of both normal and abnormal type.

The author further compares his observations with those of other investigators, and comes to the following conclusions on the basis of the reports of about 6,000 cases: (1) the type of physique originally described by Kretschmer as characteristic of schizophrenia is found just as frequently among normal persons; (2) the pyknic type of physique, on the other hand, shows a definite relation to cyclothymic character and manic-depressive psychoses;

(3) a rather loose and unreliable relation seems to exist between the leptosome-athletic-dysplastic type and schizophrenia.

Comparison between normal persons and criminals shows only one point of importance: the much smaller number of pyknic persons among criminals. The type of crime, too, seems to show a certain relation to the reaction type of the criminal (cyclothymic or schizoid). Thus, cyclothymic persons commit less of the vagabond and thief type of crimes but more of the affect crimes (murder, etc.) and incendiarism. This seems to be the most important fact in the study of criminals as a constitutional group. The fact that schizophrenic psychoses, as well as schizoid persons, form the major portion of criminals can be explained by the greater proportion of schizoid types among normal persons, and by the fact that the schizoid make-up is one that renders adaptation more difficult.

MALAMUD, Foxborough, Mass.

THE REACTIONS OF AMBLYSTOMA EMBRYOS FOLLOWING PROLONGED TREATMENT WITH CHLORETONE. S. A. MATTHEWS and S. R. DETWILER, J. Exper. Zool. **45**:279 (May 5) 1926.

Amblystoma embryos of different stages were placed in chloretone solutions (1:3,000, 1:2,500 and 1:2,000), which gave complete anesthesia, and were left there during the period when the swimming mechanism normally develops. At intervals the embryos were removed and their behavior and their reactions to stimuli and movements were studied. In short periods of time following the removal to water, the normal sequence of reactions was elicited. When the embryos are anesthetized for longer periods (from ten to thirteen days) the initial reactions are usually abnormal, but recovery of normal responses gradually follows, especially if the weaker concentrations of the anesthetic have been used. Narcosis for periods longer than thirteen days is followed in all cases by atypical and feeble responses, which are never corrected. Evidently chloretone, when used for short periods (up to seven or eight days), has no obvious effect other than to retard development. More prolonged treatment injures the reflex mechanism in some specific way. Recovery of normal responses depends on the concentration of the anesthetic as well as on the duration of anesthetization. The question arises whether actual function is necessary for the completion of differentiation of reflex arcs. When the question is considered in the light of the work of other investigators, it seems that we must look to more elementary processes, such as the metabolism of growth, rather than to functional activity for the causes underlying the orderly sequence in the development of normal nervous pathways.

WYMAN, Boston.

THE BIOCHEMIC FACTOR OF AUTOINTOXICATION IN THE CAUSATION OF HYSTERIA. GEROLAMO CUNEO, Note e Riv. di psichiat. Pesaro, 1923, vol. 2.

Cuneo studied the case of a girl, aged 22, who had frequent paroxysmal attacks of hysteria. With an intake of 16 Gm. of nitrogen, the excretion totaled only 6 Gm. Cuneo believes that, with a retention of from 9 to 10 Gm. of nitrogen daily, the functions of different organs are bound to become affected through the production of abnormal toxic products. In the treatment of such a patient, a diet intended to bring about equilibrium between ingested and excreted nitrogen is of the utmost importance. The patient, while in the hospital, was given a carefully regulated diet, and rapidly recovered; on returning home she became careless with the diet and the former symptoms reappeared. On again instituting a proper diet, she began to improve promptly.

This is not contrary to the prevailing theories that hysteria is of emotional or psychic origin. Psychic disorders can readily produce alteration of function of many organs resulting in endocrine, menstrual, lactational and other disturbances. It is therefore readily acceptable that in hysteria there may be two factors, one psychologic and the other an autointoxication. Because hysteria has been classified under functional disorders of the central nervous system, one must not lose sight of the importance of autointoxication as a cause; no treatment of this condition can disregard this fact.

GILIBERTI, New York.

TARANTULA VERSUS TARANTULA-HAWK: A STUDY IN INSTINCT. ALEXANDER PETRUNKEVITCH, J. Exper. Zool. **45**:367 (July 5) 1926.

Pepsis marginata, a digger-wasp of Porto Rico, feeds her larvae exclusively on *Cyrtopholis portoricae*, a common tarantula. In nature the wasp finds the burrow of the tarantula, guided by sight. In captivity she finds her prey in a similar manner, but recognizes the species through touch. The time of attack depends on the condition of the ovary and does not occur until the wasp is ready to lay an egg. Digging of the "grave" may precede or follow the attack. In attacking, the wasp grasps the tarantula with her mandibles and introduces her sting between the sternum and the carapace, the point of puncture depending on the relative position of the combatants. Having paralyzed the tarantula and cleaned herself, the wasp licks the blood oozing from the wound, drags the tarantula into the "grave," lays an egg, and seals the mouth of the "grave" with soil. *Cyrtopholis* does not react toward *Pepsis* as toward other insects. She permits the wasp to touch her — even to climb over or under her — and tries to defend herself only after her leg has been grasped. In nature, apparently the only defense for the tarantula is to reach the bottom of her burrow before she has been stung, since then the abdomen cannot be pierced as it leaves no passage between it and the burrow walls. The paralysis affects the entire body and the heart stops. Nevertheless, gradual recovery takes place, though escape from the grave is impossible, because of the tight packing of the soil and the consequent impossibility to use the limbs.

WYMAN, Boston.

MANIC-DEPRESSIVE PSYCHOSES IN WHICH NO AMMONIAC AUTOINTOXICATION DUE TO INSUFFICIENCY OF THE UREOGENIC FUNCTION IS FOUND. GEROLAMO CUNEO, Riv. sperim. di freniatria, vol. 41, no. 3.

In 1914, Cuneo published the results in a case of manic-depressive psychosis studied carefully from the biochemic standpoint. He showed that the outbreaks bore a constant relation to certain characteristic biochemic disturbances. The depressive phase was associated with more or less marked retention of nitrogen. If a diet rich in nitrogen was continued, a condition of deficiency of the urea-forming function resulted, which was followed by the sudden onset of a stage of excitement; during this, Cuneo found an ammoniac autointoxication due to the circulation of ammonium carbonate caused by failure of the liver to transform it into urea. When the diet was regulated as soon as retention of nitrogen was noted, the patient remained normal. Three other patients, studied in 1915, did not show these characteristics, so Cuneo was forced to the conclusion that there must be different causes for the many conditions that are grouped clinically as manic-depressive psychosis. Symptomatologically these patients were different; they showed no disturbance of the ureogenic function and consequently no ammoniac autointoxication.

GILIBERTI, New York.

THE CONTROL OF MELANOPHORE ACTIVITY IN FUNDULUS. ARTHUR SCOTT GILSON, J. Exper. Zool. **45**:457 (July 5) 1926.

An investigation was made by pharmacologic methods of the control of the dermal melanophores or black pigment cells in *Fundulus heteroclitus*. Substances causing depression of the central or sympathetic nervous systems produce darkening of the fish, the result of expansion of the melanophores. This condition may be brought about by small doses of anesthetics, such as ether or chloretone, or by toxic doses of alkaloids, such as atropine, physostigmine, and cocaine. Epinephrine, as has long been known, produces paling of the fish by a contraction of the melanophores through its effect on the sympathetic system. The reflex paling of fish in response to light is intensified by the use of small doses of substances having a stimulating effect on the central or sympathetic nervous systems. It is thought that small doses of cocaine and of physostigmine act in this way and thus cause an increased paling of the fish. No good evidence was found to indicate a parasympathetic innervation which causes an expansion of the melanophores. Contraction of the dermal melanophores in *Fundulus* is supposed to occur as a result of a reflex mediated through the eye and sympathetic nervous system. This is supplemented by the suprarenal-sympathetic mechanism such as was demonstrated in *Phrynosoma*, the horned toad, by Redfield. Melanophore expansion as a response to black is thought to result from a central depression or inhibition of this reflex.

WYMAN, Boston.

MEASUREMENTS OF INTERPUPILLARY DISTANCES. BECKER, Arch. f. Psychiat. u. Neurol. **77**:317 (May) 1926.

The significance of abnormal interpupillary distances as a stigma of degeneracy is discussed from a psychiatric point of view. The distance referred to is that between the centers of the pupils when the eyes are fixed for parallel rays. Ophthalmologists and optometrists consider 62 to 66 mm. as the average. The higher values (sometimes even up to 78 mm.) are found fairly frequently in normal persons, in whom, however, they are proportional to the size of the skull. The author reports cases of abnormally large distances with a small skull, as well as abnormally low distances (47.5 mm.) in skulls otherwise not markedly smaller than normal. The cases quoted are in psychotic patients, and the author believes that an abnormal interpupillary distance is a diagnostically important stigma. According to the author, low figures, with skull dimensions similar to those in the psychotic patients are conclusive and can be relied on with greater certainty than skull measurements. No definite conclusions are drawn, but the author suggests the desirability of paying more attention to this sign in psychiatric material.

MALAMUD, Foxborough, Mass.

EMOTION AND THE INCIDENCE OF DISEASE. GEORGE M. STRATTON, J. Abnorm. Psychol. **21**:19 (April) 1926.

Individuals who have experienced disease have slightly less control of anger than those in whose past there has been no such weakening element.

HAMILL, Chicago.

Society Transactions

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LOCAL COOLING TESTS IN NORMAL PERSONS. THEIR MODIFICATIONS IN PATHOLOGIC STATES AND BY CARDIAC POISONS. DR. NERI.

The test developed by Neri consists of plunging the two hands into ice water for five minutes and studying the changes in temperature thereafter. In 10 per cent of apparently normal persons the return to normal temperature is slow; in soldiers who had suffered from frostbite this sluggishness in the subsequent rise was found in 50 per cent. One of these men, on plunging the hands into ice water, showed generalized effects such as pallor, yawning and sweating. A few patients showed retraction of the eyeballs during immersion of the hands. Even light compression of the limbs for a few hours caused lowering of thermic reaction. Immobility for a certain time likewise delayed the response. In cases of causalgia and sympathetic paralysis, the reaction on removal of the hands was exaggerated. Atropine depressed the reaction. Thyroid extract exaggerated it.

CLINICAL EXAMINATION OF THE VEGETATIVE SYSTEM. DR. TINEL.

The complexity and obscurity in the interpretation of the results of exploration of the sympathetic system result in large measure from too simple a conception of the vegetative system. It is customary to speak of the sympathetic and parasympathetic systems as contrasting with each other, but in reality there are several sympathetic systems and several parasympathetic systems, and the action of the parallel systems is not always identical. Sympathetic excitation may be found in the cervical region and the reverse in the trunk or limbs. A bulbar parasympathetic system is likewise often in disharmony with the parasympathetic system of the abdomen and pelvis. In addition to that there is the peripheral vegetative system, comprising cardiac and pulmonary plexuses, and pilomotor and sudoriferous mechanisms. These local peripheral systems possess a certain amount of autonomy. All known methods of exploration therefore act on the two main divisions of the involuntary nervous system and also directly or indirectly on the peripheral systems, determining in each of them reactions varying in nature. All that can be spoken of, therefore, is a general tendency in one way or another, and in the clinic all that can be done is to try to determine the general tendency of the regulatory systems at that particular time. The oculocardiac and solar reflexes probably will be found of value in clinical medicine. Several tracings are shown from various persons comparing the effect of the solar and oculocardiac reflexes on the apex impulse. The conclusion is that: in exophthalmic goiter there is great exaggeration of the tonus in both sympathetic and parasympathetic systems. In "emotional neurosis resembling exophthalmic goiter," there is greater sympathicotonus (solar reflex) without an oculocardiac response. Likewise, in Raynaud's disease of emotional origin the same characteristics were present. Treatment with pituitary extract

aroused parasympathetic tonus, brought about exaggeration of oculocardiac reflex and induced recovery of the patient. In some cases of epilepsy, there is habitual hypervagotonia which is exaggerated as the crisis approaches and disappears later. In maniacal excitement, hypervagotonia is almost the rule; in the depressions there is practically no oculocardiac reflex at times. Conservation of some vagal excitability is a favorable sign in cases of acute depression. In spite of all the reserves that must be made in such a subject, there is a definite interest and importance. Therefore, if patients can be studied without demanding any more than an indication of the general tendency of the regulatory system, help can sometimes be obtained not only in diagnosis and prognosis, but also often in therapeutic indications.

Physiologic Ideas on Vagotonia (Pancreas and Vagal Tonus). Dr. Santenoise.

Vagotonic persons have a remarkable tolerance for carbohydrates; on the other hand, those with hypotonia have a low tolerance. In patients in whom vagotonia fluctuates, there are parallel fluctuations in carbohydrate tolerance. The injection of insulin seems to have a stimulating effect on the pneumogastric, analogous to the action of epinephrine on the sympathetic system. Following injections of insulin there is slowing of the heart, exaggeration of respiratory arrhythmia, increase in the amplitude of contraction due to the lengthening of the diastolic phase and exaggeration of the oculocardiac reflex. This vagotonic action of insulin is immediate and is not modified by raising the sugar in the blood by injection of glucose, showing that insulin is a specific excitant for the parasympathetic system. In experimental animals the vagal tonus seems to run parallel with the size and weight of the pancreas, animals having large glands being largely vagotonic. In the circulating blood there is a hormone that exerts this vagotonic action; the serum coming from the pancreatic vein is extremely active. Blood drawn two hours after pancreatectomy is inactive.

Sympathetic Symptoms of Juxtavertebral Cervicothoracic Tumors. Dr. Egas Moniz.

Moniz describes the case of a man, aged 35, who complained of pains in the right upper quadrant of the chest for two years before consultation. During the following year the pain radiated to the internal portion of the right arm and later to the hand. Shortly before admission, crises of pain of great intensity occurred in the region served by the first and second roots of the right side. There were no signs of involvement of the pyramidal tract and the tendon reflexes were normal throughout. There was pain on pressure above the clavicle. Sensation to pain and temperature was greatly disturbed in the area on the inner side of the arm and across the chest and back in the regions of the first to the fourth thoracic roots. A roentgenogram of the vertebral column showed erosion and disappearance of the base of the second rib on the right side due to sarcoma. Studies of the sympathetic system revealed a clear-cut Horner syndrome on the right. The skin on the right side was drier than normal and did not react as prominently to sudorifics. The patient first complained of cold in the right hand, but later noticed that it was warmer than the other hand and that he had to hold cold objects in his hand to keep it cool. There was a difference of 2 degrees between the two sides, but the axillary temperature showed practically no difference. The right ear was redder and hotter than the left. The temperature of the face was higher on the right. The tempera-

ture beneath the tongue taken simultaneously on both sides revealed a difference of more than half a degree centigrade. The ocular and palpebral conjunctivae were injected on the right. The pilomotor reflex was more marked on the left. The skin of the shoulder and face on the right side showed no reflex. When a finger was snapped against the ear, the patient felt an electric shock in the fourth intercostal space. The presence of sympathetic without pyramidal involvement speaks strongly for an extravertebral origin of the disorder.

LESIONS OF THE SYMPATHETIC CELLS IN THE CHRONIC PSYCHOSES. DR. DIDE.

In old cases of dementia praecox, ranging from thirteen to forty years in duration, atrophic cells with proliferated capsules and swollen processes were found in the sympathetic ganglia. The majority of these cells, after having become filled by large lipoid granules, took on a skeleton-like appearance.

PHYSIOPATHIC DISORDERS. DR. FROMENT.

Two conceptions have hitherto prevailed in the field of physiopathic or reflex disorders of the sympathetic system. One invoked reflex excitation of the bulbospinal centers of the sympathetic system. The other laid the trouble to the immobility brought about by pithiatism, faulty utilization of the limb and a peculiar blood flow, manifested particularly by a small pulse. Albert of Liège has recently brought forward new evidence in regard to the causation of these physiopathic disorders. Every injury to the limbs provokes local vasomotor disorders. They develop immediately because of the simple axon reflex consequent on peripheral excitation. The first symptom that follows vasomotor disorders is muscular hypotonia, which comes on soon after the injury. The patients have difficulty in moving their limbs even though the muscles far from the site of the injury are still powerful. These reactions persist after injection of curare and even after high section or extirpation of the spinal cord. This, particularly, proves their local nature.

ORTHOSTASIS AND THE INTRAVENOUS INJECTION OF ATROPINE. DRS. LAIGNEL-LAVASTINE and LARGEAU.

A test developed by Daniélopolu consists of taking the pulse rate and the blood pressure at one-fourth minute intervals and injecting at from ten to fifteen minute intervals the following doses of atropine sulphate until the vagus is paralyzed: 0.5 mg., 0.5 mg., 0.25 mg., 0.75 mg. Paralysis of the vagus is complete when the pulse rate while the patient is standing is not above the pulse rate while he is lying down. The difference between the primary reading and the maximum acceleration indicates the inhibitory power of the vagus. This will often give a delicate indication of the tonus of the parasympathetic system.

CERVICOTHORACIC SYMPATHETIC MODIFICATIONS DURING THERAPEUTIC PNEUMOTHORAX. DRS. VILLARET, JUSTIN-BESANCON and CONTIADES.

Before injection of nitrogen into the thorax, the pulse was regular and the pupils were equal. The oculocardiac reflex was equal on the two sides. Two hours after the injection, there was mydriasis on the side operated on and inversion of the oculocardiac reflex though only by pressure on the eyeball on the same side. The pilomotor reaction occurred earlier and more sharply on the side operated on. Such phenomena as were seen in this patient are more pronounced when apical adhesions are present.

HIGH LESIONS OF THE SPINAL CORD AND PARALYSIS OF THE COLON DUE TO SYMPATHETIC HYPERFUNCTION. DR. FELTKAMP.

Cases of transverse lesion or syringomyelia in the upper part of the spinal cord occur which are characterized by atony and dilatation of the colon and rectum. The medullary centers influencing the large intestine remain intact, the sympathetic and parasympathetic systems being equally active. In general, the sympathetic predominates over the parasympathetic in the intestines. The atony is explained by the hypothesis that once the higher centers cease functioning because of a high lesion, the lower part of the spinal cord takes on automatic activity with consequent interruption of the normal movements. When the lumbar cord is injured, ileus is never produced.

FREEMAN, Washington, D. C.

SCANDINAVIAN NEUROLOGIC CONGRESS

Third Meeting, Oslo, Norway, Sept. 17 and 18, 1926

PROFESSOR MONRAD-KROHN, *President, in the Chair*

The following officers were elected: president, Professor Monrad-Krohn; vice-presidents, Prof. Marcus, Hagelstam and Viggo Christiansen.

The first day of the congress was devoted to reports and communications on neurosyphilis, the subject being presented by Borberg and Saethre. Of great interest was a short communication by Dahlström, who stated that metasyphilis is exceedingly rare among patients treated according to the principles of the late Prof. Boeck of Oslo (an antimercurialist).

On the second day various topics were discussed: Gunderson (Oslo) raised the question of a possible relation of epidemic encephalitis to epidemic parotitis. Basing his conclusions on epidemiologic observations in Norway, he demonstrated the probability of a close relation between the two diseases. "Ventriculography" and "Cerebral Operations" also were discussed by Antoni, Olivercrona, Magnus and Monrad-Krohn. Professor Henschen discussed "The Physiology of Binocular Vision" and in another communication pleaded the necessity of assuming the existence of an "agraphic center." Hansen related observations of a disease which he considered identical with Huntington's chorea; he claimed that this disease had been observed and described by a local practitioner in Norway long before Huntington. Other communications were made by Hagelstam, Marcus, Wohlfahrt, Thjötta, Winther, Höglung, Wernoe, Jörgensen, Sahlgren and Nylen, Kahlmeter and Bruusgaard.

It was decided to hold the Fourth Scandinavian Neurologic Congress in Helsingfors, in 1929.

Book Reviews

NERVOUS AND MENTAL DISORDERS FROM BIRTH THROUGH ADOLESCENCE. By B. SACHS, M.D., Consulting Neurologist to the Mount Sinai and Montefiore Hospitals; Formerly Alienist and Neurologist to Bellevue Hospital, New York, and LOUIS HAUSMAN, M.D., Instructor in Neuro-Anatomy, Cornell University Medical College; Adjunct Neurologist, Mount Sinai Hospital, New York. Price, $10.00. Pp. 861, with 110 Illustrations, Including Three Full Page Plates (one in color). New York: Paul B. Hoeber, 1926.

"This is a new book," not a mere revision of Nervous Diseases of Children by Sachs, the second and last edition of which appeared more than twenty years ago and which was a hardy pioneer in this field. The present work is not only comprehensive but aspires to be up to date. In this it is successful, although some archaic fragments may be found here and there.

Chapters I and II (sixty pages by Hausman) are on the structure of the nervous system in its relation to function and are exceedingly well done. Indeed, I know of no better succinct statement of how the nervous system is built and what it does than these two chapters. Chapter III on methods of examination is practical and contains all that is necessary. With chapter IV begins a consideration of organic diseases of the nervous system. Anterior poliomyelitis, the various forms of meningitis and epidemic encephalitis are adequately considered, the clinical descriptions being especially good.

The chapter on Infantile Cerebral Palsies (twenty-seven pages) is especially good, for Dr. Sachs has been actively interested in this subject for many years. Another exceedingly good chapter (X) deals with syphilis of the nervous system (thirty-seven pages). It is clear, reasonable and safe, and contains sufficient detail for the student and practitioner. The title of chapter XI, "Tubercular Affections of the Nervous System," is a misnomer, as this chapter is devoted entirely to compression of the cord in spinal caries: tuberculosis of the vertebrae. Tuberculous affections of the nervous system (meningitis, tuberculoma) are discussed elsewhere, but the chapter is excellent.

About eighty-seven pages are devoted to "hereditary and family affections," the muscular atrophies and dystrophies and allied or similar diseases. Just why the muscular dystrophies are not included under "hereditary and family" is not explained. Naturally, one finds the exposition of Tay-Sachs disease unexcelled. It leaves nothing to be desired. The other diseases in this group are clearly presented. Chapter XV is an excellent summary of what is known of diseases of the corpus striatum, and the following one is devoted to chorea and choreiform diseases. The part on chorea is clear, sound and sensible, but in view of the fact that tic is much more frequent than chorea in children and that it is often mistaken and treated for chorea, one could wish that this annoying disorder had received more detailed attention. The discussion of Huntington's chorea, which is exclusively a disease of adults, contains more than 1,300 words, while the discussion on tics is limited to 325 words.

The subject of tumors of the spinal cord is elaborately and well treated, and syringomyelia receives considerable attention although it almost never occurs in children. Quite properly, tumors of the brain are fully described and discussed, for "intracranial neoplasms are very common in childhood." Brain abscess also

is common in childhood and early youth and is adequately considered, as is sinus thrombosis.

To eclampsia infantum and epilepsy are devoted thirty-two pages containing the current information on these subjects. This information is reliable, and matters of opinion are handled with good judgment. But why, in a book for students and practitioners, should more than 900 words be wasted on "theories of the disease" (epilepsy) — some obsolete, some foolish and none conclusive — and exactly 124 words be devoted to petit mal, which is encountered by every practitioner and recognized by few?

An interesting and useful chapter is that on speech and speech disorders. It is to be hoped that many physicians will read it carefully.

The chapter on disorders of sleep will be found helpful, and that on disorders of internal secretion is adequate although containing a few inconsistencies of statement. Clinical descriptions of the vasomotor and trophoneuroses are good, but the pathologic condition in these disorders is treated in rather vague style.

The caption of part five (five chapters, 112 pages, for which Dr. Sachs holds himself alone responsible) is "Mental Conditions," and the first chapter is on "the normal child, the normal youth — development and training." The first pages are probably excellent for the parent but rather platitudinous for the physician. There follows an earnest, perhaps one should say fervid or even perfervid, protest against the tenets and practice of the freudians. The author's arguments are perhaps more emphatic than closely logical but obviously are based on large experience, repeated consideration and absolute sincerity. He supports his position with the opinions of eminent psychologists, neurologists and psychiatrists; and his presentation may be considered as representative of the attitude of many antifreudians. In his vigorous protest against lay persons dabbling in and practicing psychanalysis the author assuredly has the whole-hearted support of all properly qualified physicians. These pages can be commended to any one interested in the subject whether his inclinations are pro or con. That the opinions and feelings of any considerable group of neurologists should be harmonious on this subject is beyond the fondest dream of the most ardent pacifist.

The concluding eighty pages on the neuroses, psychoneuroses and insanity constitute a workable outline for the student and general practitioner.

An excellent feature of this book is a well selected bibliography at the end of each chapter, making it easy for an interested reader to pursue the subject further. The index is fair, the illustrations as a rule good, the tables helpful and the work of the publisher excellent.

SPEECH TRAINING FOR CHILDREN. By MARGARET GRAY BLANTON and SMILEY BLANTON, B.S., M.D. Price, $1.35. Pp. 261. New York: The Century Company, 1919; second printing, 1924.

The Blantons are among the first in this country to attack the problem of functional speech disorders from a psychologic standpoint. The key to the speech problem is well expressed in the seventh chapter: "In any study of the defects of speech the individual person, rather than the defect itself, must be considered, for not only do the symptoms in each case differ, but the underlying causes and the individual temperament as well. . . . The whole mental fabric of the stutterer is askew, and only those methods of treatment are of permanent value that aim at the fundamental failing and unearth the trouble lying at its root. Then may be undertaken the reeducational work necessary to alleviating the symptom." Speech may be conceived of as a "test of the psychic

adjustment of the individual person to the conditions under which he must live. It is the medium in which he presents himself to the trained listener and says: 'Here am I. This is how I meet conditions. This is what I have developed, what breadth, what depth. Here are my aspirations, my unsleeping desires. This is what my environment has made of me, and I of it." Speech cannot be considered as an entity separate from the personality, a part of a person's make-up, for it is an expression of the personality and reflects in its very character the whole personality and its adaptations. It mirrors the psychic and emotional life of the individual, whether he is well or poorly adjusted to his social sphere.

Stammering is to be regarded principally as an aberration of the personality in its relation to the social environment, and not as a disturbance of the speech mechanism itself; and on this fundamental conception is based our present day understanding and treatment of the great majority of such speech difficulties.

The book is divided into two parts. The chapters of the first part are concerned with speech hygiene and the development of the child and his expression through speech. Chapter 2 goes into a short discussion of speech training and general education of the child, with special reference to the four periods of speech development: (1) the first five years, in which the child normally acquires speech; (2) from 5 to 9 years, during which speech habits are rather firmly set; (3) from 9 to 14, a "period of readjustment" and of "great psychic danger to the individual," a time when speech correction becomes more difficult; (4) from 15 to 20, the "last period of grace," years approaching adulthood when speech correction is effected only with extreme difficulty. This chapter also discusses coordination and control of the child's voice as to loudness, emotional expression and intensity; the importance of practice and habit formation in developing speech; selfconsciousness, and the proper approach and technic for influencing a change in habits of speech. The third chapter consists of a well presented description of the mechanisms of speech. The part that each of the speech organs plays in producing sound and the coordination of all toward a unified function in speaking are well set forth. The following chapter, "The Plastic Period," considers the various needs of the infant and how they can be most healthfully met. There are sections devoted to physical and emotional needs, the care of the baby, first questions of sex, discipline in the home, and the principal of sublimation. The problems of reality versus the childish day-dream world and of normal efficient adaptation to life are briefly discussed. Chapter 6 is concerned with the developing speech needs and the hygiene of good speech. The training of the "speech faculty" is divided into four phases: (1) period of reflex and instinctive cries; (2) period of language sounds without an association of ideas; (3) period of language sounds with an association of ideas and (4) period of sentence building. It is emphasized that the serious and subtle dangers to speech occur during the transition period between the period of meaningless speech, through the acquisition of conscious control of speech, to the period of automatic control.

Unhealthy types of speech reaction are discussed, including lisping, monotonous voice, indistinct speech, lack of speech, and also the neuroses in relation to speech disorders. Chapter 9 concludes the first part of the book and takes up extra-school activities, such as social duties, platform reading, fiction reading, home study, piano practice, and arts and crafts and sinistrality.

Part II, about a third of the book, is devoted to school life and a large number of speech exercises and readings for those particular children who

require practice in enunciation and expression. The exercises include use of coarse and fine muscles, pronunciation, letter position, inflection, reading aloud, tone, resonance, emotional expression and relaxation.

Since the book is intended especially for the use of parents and teachers, who often lack background for treating speech defects, it seems that it would have been of value to include case histories with treatment, illustrative of the various conditions and difficulties mentioned in part I. This volume is the outgrowth of years of observation and experience with speech cases, and is one of the first books to represent a sound and scientific approach to the speech problem, with the emphasis rightly placed on the psychologic side. The book is of unquestioned value to all those interested in functional as well as physical disorders of speech.

UEBER DIE VERWAHRLOSUNG DER JUGENDLICHEN. By W. RUNGE and O. REHM. Monograph 37 of the "Abhandlungen aus der Neurologie, Psychiatrie, Psychologie und ihren Grenzgebieten, Beihefte zur Monatsschrift für Psychiatrie und Neurologie" edited by K. BONHOEFFER. Price, 7.50 marks. Pp. 156. Berlin: S. Karger, 1926.

The problem of the young delinquent is approached by the authors from two points of view, the clinical and the social. Runge presents the clinical side of the question. His investigation is based on a study of the German literature and of a fairly large material of his own, consisting of cases of young delinquents observed in an outpatient clinic. It is more a comparative and statistical than a purely clinical study of the material. The basic principle is that, although environmental influences materially aid in precipitating delinquency, the major cause is constitutional. The constitutional factors consist mostly in abnormal psychic make-up, and investigation brings out the following proportional representation of such abnormal states: the most frequent is psychopathic personality; the next in frequency is feeblemindedness; organic disease (encephalitis, etc.), epilepsy and incipient or abortive psychoses are represented to a much less extent.

Given an abnormal psychic state, delinquency may be brought about even when environmental conditions are not particularly difficult to cope with; although, in a bad milieu, delinquency will be precipitated much more frequently and even when the constitutional factor alone might not, of itself, have been sufficient cause. A comparative study of delinquent and nondelinquent "abnormals" brings out the following points: On the whole, hereditary factors seem to influence delinquent abnormal persons more than socially adapted abnormal persons, this being more true in psychopathic than in other types of abnormality, and least of all in feebleminded persons. Certain factors in heredity, especially alcoholism and sexual discrepancies, are particularly conducive to delinquency, though here one has to consider the environmental influence of such factors. Some psychopathologic features seem especially to favor the development of delinquency; these are aggressiveness, cruelty, hypersexualism or perverse sexuality, fickleness, etc. On the other hand, there are pathologic features which help to keep the individual from becoming delinquent: "nervousness," shyness, prudishness, the stronger inhibitions of psychoneuroses, etc. The type of delinquency seems to be influenced by the age and sex of the person. The proportion of sexual delinquency is more pronounced in children below 14 and in the female, whereas stealing, running away, vagabondage, etc., are more frequent in older children and in the male.

Rehm discusses the social aspects of delinquency. He, too, is of the opinion that constitution plays a much more important rôle than purely environmental

factors. His figures are: purely active (a term used to cover the constitutional type), 50 per cent; purely passive (milieu), 7 per cent; the rest is a mixture of both passive and active. The evaluation of social factors, as well as suggestions as to the possibility of preventing delinquency, are strongly colored by this attitude and observations. In the foreground of prevention, we find eugenics. Sterilization, castration and other measures to stem hereditary transmission are of the greatest importance. In addition, however, one should attempt to improve such elements in environment that are liable to act as precipitating or even causative factors. Both authors consider organic intoxications as of secondary importance. Syphilis and alcohol and other drugs are much more the result than the cause of delinquency, although they no doubt aggravate it.

The work, especially the first part, as stated, is largely comparative and statistical. The bibliography given and considered is confined to a special sector of the German literature, and one misses the discussion of other views on the subject, especially those of American workers. Much of the work on the deeper psychologic mechanisms that may be responsible for delinquencies is not considered. Both in the personal material of the authors and in the study of work done by others, the discussion of possible environmental influences deals more with descriptive and static than with dynamic values.

As an exhaustive study of the problems of delinquency the monograph leaves much to be desired. The work will prove of interest and value to students of the subject, provided they realize that only certain aspects are dealt with. The literature that is considered is discussed thoroughly and the attitude of the authors to their own material, that of others, and the subject in general, is stated clearly and as definitely as is possible.

UEBER DIE AGRAPHIE UND IHRE LOKAL DIAGNOSTISCHEN BEZIEHUNGEN. By DR. GEORG HERRMANN and PROF. DR. OTTO PÖTZL. Price, M. 24. Pp. 380, with twenty-nine illustrations. Berlin: S. Karger, 1926.

This volume describes a single case in which agraphia is the predominant symptom. In the detailed presentation, there is general agreement with the recently expressed views of Henschen combating the idea, by no means new, that agraphia is not an entity. A praiseworthy attempt has been made to avoid confusion between psychologic and physiologic points of view and to develop a parallelism in methods of approach without falling into the error of psychologic localization.

The contents of the book may be summarized as follows: (1) return of agraphia following extirpation of a parieto-occipital tumor; (2) the relationship of agraphia to apraxia, to word blindness and to aphasic disturbances; (3) disturbances of position in agraphia; (4) relation between the central agraphic region and the motor areas; (5) comparative consideration of cortical lesions in the cases of pure agraphia hitherto observed; (6) right-sided and left-sided lesions in persons with agraphia; (7) effects on the hands in writing, and (8) necropsy observations in individual cases. The second part of the book is concerned with an elaborate discussion of the literature relative to central disturbances of the capacity of writing, with a somewhat detailed statement of the views of Bastian, Wernicke, von Monakow, Pick, Henschen and others.

The case about which the investigation is built concerned a man, aged 49, who gradually developed signs of brain tumor, ultimately localized in the parieto-occipital region. Operation resulted in the removal of the growth, which, however, was not absolutely complete. Consequently, there was a recurrence with a reappearance of agraphia, which for the time being had

been relieved by the removal of the tumor. Necropsy further substantiated the operation in determining the location of the tumor, as was predicated by the clinical examination. Throughout, the essential symptom was agraphia. Operation showed that the tumor lay in the transitional region between the second occipital convolution and the angular gyrus, and was presumably malignant, with rapidly increasing pressure toward the base of the brain and the splenium of the callosum, as was indicated by the clinical observations. The fact that the tumor was on the right side of the brain and that the aphasia with predominant agraphia resulted therefrom is explained on the basis of ambidexterity of the patient. The coincidence of parietal alexia and parietal agraphia in the case described constitutes a definite syndrome according to the authors, which is to be distinguished from pure word-blindness as well as from aphasia in general and demands special study. A careful investigation is made relative to the relationships of the central agraphic region with the different motor systems, and in general it may be said that, somewhat on the basis of the older conceptions of aphasia, the authors have again gone into the most extreme details regarding many matters, which in last analysis must still be regarded as theoretic. The old discussion regarding the identity of an agraphic area and its relation to the prefrontal region as described in Gordinier's well-known case is reopened, and the fact that this area, in close relation to the so-called arm center, cannot be regarded as the sole area of writing is established.

The entire book is a characteristic, painstaking investigation, on the basis of small personal material, of the always difficult problem of aphasia. No doubt the sum total of cases thus carefully analyzed will add greatly to our ultimate knowledge, but there is always danger, which possibly may be urged as a criticism of this work, that generalizations should not yet be drawn, on the basis of observations so far available. The whole tendency toward simplification and reorganization of our knowledge of the aphasia problem, which has been so apparent in the last few years, is perhaps not forwarded by an investigation of this sort. It is, however, a valuable contribution to a discussion of previous theories and places on record a carefully localized and studied case of agraphia, amply verified both by operation and by subsequent necropsy. For students of the general problem, the book will be of great value. It can, however, hardly be read with profit by those uninitiated in the subject.

MEDIZINISCHE PSYCHOLOGIE. By E. KRETSCHMER. Price, 15.30 marks (bound, 17.50). Pp. 273. Leipzig: Georg Thieme, 1926.

The third edition of the "Medical Psychology" has been materially enlarged. The groundwork of a conservative Wundt-Kraepelin type is the same as in the previous editions. Into this, however, the author attempts to incorporate the results of more recent investigations of organic, psychologic and clinical research. The recent works of Freud, Schilder, Adler and Jaensch on the one hand, as well as of Vogt, Förster, Goldstein and Head, of the organic school, on the other, are discussed in an impartial, even if cursory, manner. Considering the space and the amount of information, the presentation is exceptionally clear and easy to follow.

Is a medical psychology possible? The answer to this question would logically depend on the peculiar relation between the two concepts, medicine and psychology. Presumably it is neither a presentation of psychologic investigation nor a text-book on psychotherapy that one would expect. It is rather the relation between

the two, or to be more exact, the understanding of psychotherapeutic measures in the light of psychologic investigations. Beginning with the presentation of current, well established psychologic views as the central point, and touching here and there on some extremes in the field, the author attempts to take us through the turbulent waters of the study of human nature. The various mechanisms of normal and abnormal behavior are built up on a developmental plan, and with the aid of compromises here and there, some of the less approved or not as yet definitely established theories are built into the older ones.

At the end of the book is a chapter devoted to psychotherapy. Here, however, the author changes his attitude completely. Scientific investigation is all very well for theoretical considerations. Psychotherapeutic measures, on the other hand, seem to be more of an art than a science. "Magic is the most primitive form of psychotherapy," and the author, true to his developmental plan of study, presents the modern psychotherapist as the direct successor of the original medicine man. Thus, having read through the book, one seems to find even more of a gap between practical psychotherapy and evaluation of psychopathology, on the one hand, and theoretical psychologic considerations, on the other, than one had before.

The presentation of the psychologic material and the discussion of modern trends, however, are extremely interesting and whether one agrees with the author's views or not, should prove valuable to the student of the subject.

EPILEPSY: A FUNCTIONAL MENTAL ILLNESS. By R. G. ROWS, M.D., LONDON and W. E. BOND, M.R.C.S., ENGLAND, L.R.C.P., LONDON. Price, $3.00. Pp. 138. New York: Paul B. Hoeber, 1926.

The death of one of the authors, R. G. Rows, caused Dr. Bond to publish the manuscript as Rows left it. The conclusions arrived at are based on experiences during the war. The authors refer constantly to these patients, but there is no detailed description, so that the reader cannot judge the results and the methods by which they arrived at their conclusions. They start with the preamble that prewar conceptions of fits are not adequate, and that therefore some other explanation is necessary. "Emotional disturbance was the original cause of the first fit in the large majority of the illnesses, and the same factor instigated many of the recurrent fits." They discuss the mental make-up of the epileptic patient and state that the repetition of the emotional states and of the bodily reactions which expressed them leads to its development; they call attention to the fact that epileptic patients, like those who suffer from other forms of mental illness, must be considered individually, and that they may have any type of mental reaction. In discussing the aura, which to them is the result of the reactivation of an idea based on the original stimulus, they point out that the mood present before the fit is of some importance, for the latter might be an actual reproduction of the feeling present when the first attack of unconsciousness supervened.

It is obvious from this that the authors regard epilepsy as a mental state based on a previous emotional experience. The discussion of treatment is cursory. It is divided into three stages: explanation, exploration and reeducation. The book is suggestive, proves interesting reading and presents a point of view that should be considered by all organic neurologists in spite of the fact that many will not approve of it. The book was published in incomplete form, for some of the newer ideas of epilepsy are not incorporated, and it is lamentable that a full exposition of the cases studied was not included.

NEUROLOGIE. LES CONSULTATIONS JOURNALIÈRES. By AUGUSTE TOURNAY. Price, 20 francs. Pp. 309. Paris: Gaston Doin, 1926.

This is a pocket-sized manual designed for the physician in general practice, telling him how to make diagnoses and what to do in cases of nervous diseases, and also indicating what can be done for the relief of such patients by properly trained personnel. The emphasis is always on treatment, and it is interesting to note the increasingly large number of therapeutic methods that find application in cases of nervous disease. For the relief of pain, for example, one can start with coal-tar analgesics, proceeding to opiates, physiotherapy, electrotherapy, and radiotherapy and then resort, if necessary, to surgical procedures such as intraneural or epidural injections, rhizotomy, sympathectomy and cordotomy.

The material is unevenly stressed, much space being devoted to simple procedures while decompression, ventricular puncture and malaria inoculation are given a description that is entirely inadequate for one who would attempt them. The book is not a catalogue of prescriptions; indeed, few formulas are given, and polypharmacy is distinctly frowned on. Physiotherapeutic measures are given full weight, and minor surgical procedures (injections, etc.) are fully stressed. Diagnostic methods are sketched, and symptoms are indicated after the fashion of a compend with much italicism, so that he who runs may read. Some sections are bare statements, but others, such as that dealing with convulsive seizures, are prolix. Yet epileptic equivalents, which are of such importance in the diagnosis of epilepsy, are dealt with summarily.

The author is fairly accurate in his statements, though some authorities would deny that congenital syphilis is the cause of nearly all cases of infantile encephalopathy. He is also dogmatic in saying that every case showing symptoms of complete transection of the cord should be abandoned by the surgeon. Only a short chapter is devoted to functional nervous disorders.

As the publisher states, the book is written for the practicing physician, particularly in the "provinces," who presumably has not kept pace with refinements in diagnostic and therapeutic procedures. It is designed to lead him to differentiate hysteria from brain tumor and to take the proper course. If the book appears like an ethical type of advertising for Parisian neurologists and surgeons, it is a kind of advertising that would be appreciated in the "provinces." For the practicing neurologist, it has value in recalling therapeutic procedures that may be used in various cases of nervous disease.

HANDBUCH DER NEUROLOGIE DES OHRES. By PROF. G. ALEXANDER, PROF. DR. O. MARBURG and DR. H. BRUNNER. Volume 3. Price, 66 marks. Pp. 825. Vienna and Berlin: Urban and Schwarzenberg.

This volume marks the completion of the series that was begun several years ago. The first two books were reviewed in a previous number. The present volume begins with a chapter by Professor Marburg on tumors in the region of the cochlear vestibular system and the cerebellum. In this are included lesions of the medulla and the pons up to the corpora quadrigemina. This is undoubtedly the best short presentation of the subject to be found anywhere, for the author not only gives his own point of view but discusses freely the literature on the subject. The discussion of the pathologic changes of tumors of the posterior fossa is particularly interesting, especially in view of Cushing and Bailey's recent work on the subject, which the author did not have the opportunity to review. This excellent article is followed by the

operative treatment of brain tumors by W. Denk. There is nothing particularly striking about this chapter, except that ventriculography is given considerable attention. Then follows a discussion of the roentgen-ray treatment of diseases of the ear and of tumors.

The next subdivision is on the dyskynesias and dystonias, by Pollak of Vienna. There is an excellent discussion of tone, particularly in relation to the labyrinth. The most interesting part, however, is the relation of the vestibular apparatus to the so-called extrapyramidal system, with a good discussion of the striatal diseases including their pathologic conditions. Then follows a description of the neuroses and psychoses in which attention is directed to the prevalence of functional disturbances in the cochlear and vestibular apparatus and particularly of dizziness. This is too often overlooked by both neurologists and otologists. In this subdivision are considered also epilepsy, autogenic reflex neuroses, the psychoses associated with disorders of the ear, nystagmus, seasickness, the vegetative reflexes of the labyrinth and spasmus nutans; there is an excellent chapter on flying and its relation to the function of the eighth nerve. The final chapter is on the glandular diseases with a particular discussion of the nervous system of the ear in cretinism and myxedema.

Too much praise cannot be given the editors who have so successfully completed this work. They have given us an eminently satisfactory treatise on the nervous system of the ear which leaves nothing to be desired.

LES AFFECTIONS DE LA COLONNE VERTÉBRALE. By ANDRÉ LÉRI. Pp. 526. Paris: Masson et Cie., 1926.

Twenty-five years ago, when Pierre Marie formulated his ideas about a chronic articular disease and called it spondylose rhizomélique, André Léri was his pupil and collaborator. Since that time Léri has not ceased to study diseases of the spinal column and has made valuable contributions to the knowledge of some of these diseases. The present book is the summation of his investigations and publications. It brings the subject fairly well down to date and is perhaps the best book I know of on this subject. However, the book is written from the standpoint of the internist and the neurologist. In no sense is it a surgical work, nor can it be said that it is a systematic treatise. It is rather a collection of valuable papers on various forms of spinal disease.

The first section, 215 pages, is devoted to anomalies and disorders of development. Perhaps the most interesting chapter in this section is that on the association of spina bifida occulta and "essential" incontinence of urine. In rebellious cases of nocturnal incontinence, Léri generally finds occult spina bifida, and operation reveals an abnormal band compressing the cauda equina. Division of this band relieves or palliates the incontinence.

There are also chapters on trophedema in spina bifida, sacralization of the fifth lumbar vertebra, lumbarization of the first sacral vertebra and the cervical ribs, and the symptomatology of a deficient number of vertebrae.

Section II is a short one in which heredotraumatic kyphosis, roentgenography in latent spinal fracture and progressive postraumatic compression of the cord are discussed.

Section III, seventy-four pages, is on ankylosing diseases of the spine, and section IV, 154 pages, on localized rheumatism of the spine and its consequences. Being a loyal pupil of Pierre Marie, the author naturally separates spondylose rhizomélique from other osteo-arthritides of the spine. Whatever one's ideas

of classification may be, these two sections constitute an interesting and instructive exposition of the subject as regards both clinical manifestations and pathology. In a short chapter on treatment of these diseases by thorium X, the author claims good results from hypodermic or intramuscular injection once a week of from 100 to 400 micrograms. The remedy is unstable.

THE CLINICAL EXAMINATION OF THE NERVOUS SYSTEM. By G. H. MONRAD-KROHN. Ed. 3. Price, $2.50. Pp. 201. London: H. K. Lewis & Co.; New York: Paul B. Hoeber, 1926.

A pocket-size manual for guiding the student, the practitioner and the neurologist in the examination of the nervous system is no mean accomplishment. Monrad-Krohn details the technic necessary for eliciting various signs, with concise descriptions of the results obtained. While not a textbook of neurologic diagnosis, it is a happy combination of the most important procedures with a consideration of the localizing value of the signs elicited. The work is systematic. It begins with a discussion of cerebration; the cranial nerves, trunk and limbs are considered next; this is followed by a full consideration of sensation and reflexes. Descriptions of electrical and spinal fluid examinations follow. Not the least important is the appendix in which the psychosomatic functions are dealt with and intelligence, visual and vestibular tests are detailed. Pharmacodynamic tests are given a place, and a series of clear diagrams used in cerebral, spinal, peripheral and sympathetic localization is given. A few paragraphs at the end suggest a procedure for the first examination, and another dilates on the value of repeated examinations.

The book is concise and readable and is illustrated by photographs and original drawings. It is a mine of information brought within reasonable limits. It is no mere compend, however, the author having drawn on his own experience, and if certain sections seem a little large in proportion to their probable importance, e. g., the innervation of the face, it is easy to find the reason in the author's contributions on the subject. The book is thoroughly up to date in dealing with postural reflexes, organic reflexes, cistern puncture and pharmacodynamics. Cerebral pneumography and injections of iodized oil 40 per cent are referred to, but are not dealt with in detail since these procedures are as yet outside the domain of the practicing neurologist.

As a guide for students in the examination of patients and to refresh the memory of the practicing neurologist, it takes a high rank among books of its kind. It shows what to do to obtain all the information and leaves to larger works the full explanation of what this information means. Emphasis is laid on anatomic location rather than on pathologic differentiation, on the theory, perhaps, that if localization is perfect in all spheres, the nature of the process must become evident. This book also reveals the extraordinarily varied modes of approach to the problem of localization in nervous diseases; yet the tests are chosen with discrimination and are worked into an harmonious whole. The author, although a Norwegian, writes excellent English.

AVIATION MEDICINE. By LOUIS HOPEWELL BAUER, A.B., M.D. Price, $7.50. Pp. 241. Baltimore: The Williams and Wilkins Company, 1926.

This is probably the first book written on aviation medicine since the war, and the first one in America. It is published by the authority of the Surgeon General, United States Army, the author being a member of the Medical Corps on duty in the School of Aviation Medicine. There is nothing original in the book, but it gives an excellent summary of the problems encountered by

aviators during and since the war. The army maintained an experimental laboratory during the World War. The selection of the aviator as well as keeping him in fit condition became a serious problem. That aviation medicine encompasses practically all the specialities of medicine is shown in this work, in which there is a discussion of the relation of the eye, the ear, nose and throat, the nervous system and the respiratory and circulatory systems. To neurologists, the discussion of the so-called Bárány tests, which were used largely in the selection of aviators, and the neuropsychiatric factors are of chief interest.

STAMMERING AND ITS TREATMENT. By SAMUEL D. ROBBINS, A.B., A.M. Price, $2.00. Pp. 121. New York: G. E. Stechert & Co., 1926.

This little book was written primarily for the author's pupils and is therefore published in "pocket" form. It contains a good description of what stammering is and the theories of its etiology. Treatment is the main interest, and the author describes his own method with which he has had an extensive experience. Essentially, this consists of speaking on a smooth, relaxed expiration, prolonging and emphasizing the vowels, while the consonants are largely disregarded. There are also chapters on the mental hygiene necessarily associated with the treatment—on fear, suggestion and habits. The book is of use to physicians because it expounds a rational and natural method of treating stammering; it can be used by patients as a guide while undergoing treatment.

A MANUAL OF NORMAL PHYSICAL SIGNS. By WYNDHAM B. BLANTON, B.A., M.A., M.D. Price, $2.50. Pp. 207. St. Louis: C. V. Mosby Company, 1926.

This book is of small interest to neurologists. It gives a schematic outline of methods of examination. Examination of the nervous system is treated separately and consists of ten pages, including mental examination which takes a page and a half. It is too cursory to be of any value.

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